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Research Paper

Experimental evidence of the compressibility of arteries

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ABSTRACT

A definitive answer to the question whether artery walls are incompressible is to our opinion not yet categorically provided. Experimental-based evidence on the level of compressibility in artery walls is not easily achieved because of the difficulties associated with the measurement of very small differences in volumes under physiological pressure in these biological tissues. Past experiments aimed at addressing the question considered different species, different arteries, the experimental devices were not accurate enough and a statistical analysis of the results was missing.

A precise experimental device together with a thorough testing protocol, a careful selection of arteries and a statistical analysis is presented for a definitive evaluation of the artery wall compressibility. We provide experimental evidence that in saphenous and femoral porcine arteries under physiological pressure range a relative compressibility of 2–6% is observed. The pre-assumption of incompressibility in many phenomenological constitutive models of artery walls should probably be re-evaluated.

1. Introduction

The biomechanical response of the artery tissue is a topic of major importance and intensive research, and several phenomenological constitutive models have been proposed for the prediction of their passive response. The common models are hyperelastic (uniquely determined by a strain energy density function, SEDF, Ψ) pre-assuming the incompressibility of the artery tissue under physiological conditions, see e.g. the recent review (Holzapfel and Ogden, 2010). This assumption is based on the argumentation that the artery wall is comprised mostly of water, which is considered incompressible.

In this case, the SEDF contains only an isochoric (volume preserving) part associated with the elastic matrix, and a part associated with the two families of the collagen fibers. For example in Holzapfel et al. (2000) the following decomposition of the SEDF was suggested:

$$\Psi = \frac{\mu}{2}(\mathrm{I_C III_C}^{-1/3} - 3) + \Psi_{fibers}, \tag{1}$$

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where I_C and III_C are the first and third invariants of the right Cauchy–Green tensor **C**, respectively. The material parameter μ is associated with the shear modulus at infinitesimal strains (or ground-state).

However, if in reality the artery is compressible or even slightly compressible, the SEDF has to be enriched by another volumetric term that must account for it. One such option is Yosibash and Priel (2011)

$$\Psi = \frac{\mu}{2}(I_C III_C^{-1/3} - 3) + \frac{\kappa}{2}(III_C^{1/2} - 1)^2 + \Psi_{fibers}, \quad (2)$$

with the material parameter κ being associated with the bulk modulus at infinitesimal strains (or ground-state).

The stresses resulting from SEDFs under the assumption of incompressibility are significantly different compared to those obtained under the slightly compressible assumption (see e.g. Misra and Chakravarty, 1980). As an example, slight compressibility (volume-change of up to roughly 3%) resulted in circumferential stresses in the arterial wall up to 100% higher compared to the incompressible case for the physiological pressure of 100 mmHg as documented in Yosibash and Priel (2012) and Elad (2011). For almost-incompressible materials $\varepsilon = \mu/\kappa$ is usually a small parameter, but not zero, and the interested reader is referenced to a detailed analysis of such cases for $\varepsilon \ll 1$ in Horgan and Murphy (2009) and Horgan and Saccomandi (2003).

A definitive experimental-based answer on the level of compressibility in artery walls is therefore of biomechanical interest but not easily answered because of difficulties to measure accurately very small differences in volume under physiological pressure.

Numerous publications in the past three decades report on experiments aimed at measuring the "incompressibility" in arterial walls. The reported results, however, differ significantly due to the variation in methods, number and quality of examined arteries and lack of a systematic experimental protocol and statistical evaluation of the experimental observation.

We first provide a critical review of these past experiments: Lawton (1954) experimented on dog aorta, investigating the thermo-elastic behaviour under uniaxial loads. In part of his study he used a dilatometer to determine the volume-change of the aorta during uniaxial tension. The apparatus (shown in Fig. 1) included a brass tube in which the specimen was submerged in saline solution while measuring the change in liquid level during extension. Lawton implemented a thermodynamic theory to calculate the tension force taking the change in volume into account. A small change in volume (less than 1%) was reported being within the range of experimental error, resulting in the conclusion that the artery wall is incompressible.

Carew et al. (1968) combined analytical and experimental approaches to assess the compressibility of the artery in terms of the bulk modulus κ. Cylindrical segments of various dog arteries submersed in a control volume filled with physiological solution were tested. The arteries were stretched to 3–10% of their initial length, and inflated to pressures of 167–197 mmHg. Then the saline solution within the specimen was allowed to transfer to the glass flask, allowing the inner and outer pressures to equilibrate. The change in water level was a direct representation of the change in tissue's volume. Bulk modulus was calculated

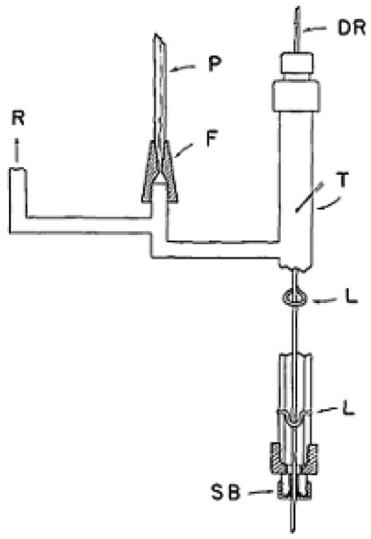

Fig. 1 – Schematic drawing of the dilatometer (Lawton, 1954). The specimen was connected to the loops (L) and the change in volume was measured by the liquid extruded into the pipet (P).

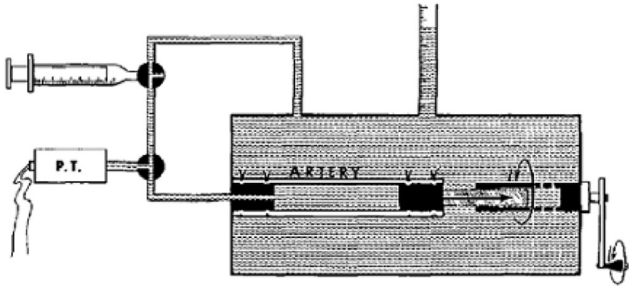

Fig. 2 – Schematic drawing of the dilatometer as shown in Carew et al. (1968). Saline was pumped into the artery, and stretched to 3–10% of its initial length. The capillary tube shown at the top was directed horizontally, thus the water leaving the glass flask through the tube did not affect the outer hydrostatic pressure.

using the axial, circumferential and radial stresses, measured by the inner pressure and axial load. The testing device is shown in Fig. 2. It was concluded that the volume-change in dog aortas is negligible, and generally under 1%.

Tickner and Sacks (1967) reported the highest volume-change in comparison with other studies, 20–35% for various human arteries. They placed the tested specimens vertically, sealed with a small weight at the bottom, designed to stretch the artery and seal it (see Fig. 3). The arteries were inflated by air and wall thickness determined by X-rays so the change in volume was computed for different inner pressures and axial loads. A decrease in volume of up to 35% for an inflation pressure of 300 mmHg was reported. The dry environment and air used for inflating the artery may had resulted in an extreme volume change.

Chuong and Fung (1984) experimented on rabbit aortas, cutting the artery open into a rectangular segment and compressing it (Fig. 4) while measuring the amount of liquid extruded from it (assumed to be the volume-change of the tissue). The compressibility for four aorta specimens was