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Initiation of aneurysms as a mechanical bifurcation phenomenon

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ABSTRACT

Recent studies on localized bulging in inflated membrane tubes have shown that the initiation pressure for the onset of localization is determined through a bifurcation condition. This kind of localization has also been shown to be much more sensitive to geometrical and material imperfections than classical sub-critical bifurcation into periodic patterns. We use these results to show that the initial formation of aneurysms in human arteries may also be modeled as a bifurcation phenomenon. This bifurcation interpretation could provide a theoretical framework under which different mechanisms leading to, or reducing the risk of, aneurysm formation can be assessed in a systematic manner. In particular, this could potentially help in assessing the integrity of aneurysm repairs.

1. Introduction

An aneurysm is a localized, blood-filled balloon-like bulge of a blood vessel. As the size of an aneurysm increases, there is an increasing risk of rupture, resulting in severe hemorrhage, other complications or even death. Aneurysm formation has broadly been associated with hereditary predisposition, old age and hypertension, and more specifically with defects in extracellular matrix maturation, increased degradation of elastin and collagen, aberrant cholesterol homeostasis, or enhanced production of angiotension peptides. However, how such factors manifest themselves in changing the mechanical behavior of arteries before aneurysm formation is still not fully understood. Existing studies in the bio-mechanical community have largely focussed on modeling the material properties of mature aneurysm tissues and the growth of aneurysms when they have already formed; see, e.g., Humphrey and Rajagopal [1], Venkatasubramaniam et al. [2], Watton et al. [3], Baek et al. [4,5], Vande Geest et al. [6], Vorp [7], Watton and Hill [8], and the references therein. Such studies are obviously important in guiding a clinician when to intervene when an aneurysm has been diagnosed. Our current study, however, is focussed on understanding the process leading to the initial formation of an aneurysm.

The geometrical similarity between a localized bulge in an inflated hyperelastic membrane tube and an arterial aneurysm is obvious, but the former problem is much better understood thanks to a large number of experimental, numerical and analytical studies, and the absence of uncertainty in the material modeling. When a hyperelastic membrane tube is inflated by pumping in air or water, a localized bulge will form when the internal pressure reaches a certain critical value. Once a bulge has been initiated, its early stage of growth is highly unstable, which takes place at decreasing pressure (and associated reduction of radius away from the center of the bulge). For almost all rubber-like materials, growth will stop when the bulge reaches a so-called *Maxwell state* which is stable. Further inflation will force the bulge to spread in both directions and will take place at a constant Maxwell pressure and constant maximum radius. The earliest documented observation of localized bulging in inflated membrane tubes seems to have been by Mallock [9]. For a selection of experimental, numerical and analytical studies, we refer to Yin [10], Chater and Hutchinson [11], Kyriakides and Chang [12,13], and Shi and Moita [14]. Although this problem has been loosely referred to as a stability/bifurcation problem, its precise stability/bifurcation nature was not fully understood until very recently; see Fu et al. [15]. In this paper, the localized bulging was recognized as a non-linear bifurcation problem (the corresponding linear bifurcation analysis incorrectly predicts the bifurcation mode as a uniform radial expansion), and it was shown that the initiation pressure may or may not equal the limiting pressure associated with uniform inflation depending on end conditions. Equality holds if, for instance, the ends are closed and any external axial force is fixed, but in the case of open ends localized bulging would occur before the limiting pressure is reached. Characterization of the entire inflation process and its stability was carried out in two subsequent papers [16,17]. In a more recent paper [18] it was further shown that the initiation of localized bulging in inflated membrane tubes is more sensitive to material and geometrical imperfections than classical sub-critical bifurcations into sinusoidal patterns. The latter obeys Koiter's

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two-thirds power rule [19,20] whereas the former obeys a square root rule. For instance, a localized wall thinning that corresponds to a 10% maximum wall thickness reduction can easily induce a 19% reduction in the critical circumferential stretch. Since for arteries the pressure is an exponential function of circumferential stretch, such a 19% reduction in stretch can reduce the critical pressure by many orders of magnitude. Such severe imperfection insensitivity has the potential to bring the critical pressure down to physiologically possible values and is crucial to our argument in the present paper.

It is well known that arteries are inelastic: they show hysteresis when subjected to cyclic loading and unloading, and also exhibit typical viscoelastic behavior (i.e. stress relaxation at a constant strain and strain creep at a constant stress), but for many practical purposes, including ours in the current paper, it suffices to model them as non-linearly elastic and anisotropic. There is, however, a major difference between the behavior of rubber-like materials and that of arteries, namely that for the former the strain-energy function can be accurately described by an algebraic function (see, e.g., Ogden [21]), whereas for the latter the behavior is typically exponential (see, e.g., Fung et al. [22]). Furthermore, arteries have a layered structure and behave anisotropically, and as a result there is much uncertainty in their mathematical modeling. Because of these differences, when aneurysm formation is interpreted as a bifurcation phenomenon it is not immediately clear whether the bifurcation condition has any solutions at all. Then to demonstrate that aneurysm formation can indeed be modeled as a bifurcation phenomenon, we need to first choose an appropriate material model, and then to verify that bifurcation cannot only take place but also can be achieved at physiologically possible pressure values. A number of material models for arteries have been proposed in recent years; we refer to Humphrey [23] and Holzapfel and Ogden [24] for comprehensive reviews. In this paper, we select two representative material models. The first is the multi-layer structural model of Holzapfel et al. [25] which also gives material parameter data for carotid arteries of a young rabbit. The second is the single-layer arterial model proposed by Choi and Vito [26] with data provided by Vande Geest et al. [6] for a group of healthy but elderly human aortic arteries. It is known that aneurysms are rare in animals and more so among young animals, and that aneurysms can be induced by pathological changes in elderly humans. Our challenge is to show that the bifurcation interpretation can indeed capture, at least qualitatively, these basic facts.

The rest of this paper is organized as follows. In the next section, we summarize Holzapfel et al.'s [25] multi-layer structural model and show that for axisymmetric deformations, provided each layer in an artery is hyperelastic, the composite artery is necessarily hyperelastic and we give the effective strain-energy function. This result paves the way for the application of Fu et al.'s [15] bifurcation condition which we briefly derive in Section 3 for completeness. We show that the bifurcation condition reduces to a determination of the zeros of the expression (3.11) by virtue of a local analysis of the ordinary differential equation (3.10). This bifurcation condition is then applied in Section 4 to the two material models mentioned in the previous paragraph. The paper is concluded with a summary and some additional remarks.

2. Governing equations

Healthy arteries are composed of three clearly defined layers: the intima (the innermost layer), the media (the middle layer) and the adventitia (the outer layer). Each layer of the arterial wall may be considered as a composite reinforced by two families of fibers arranged in symmetrical helices [27]. With incompressibility assumed, the strain-energy function Ψ for each layer is a function of the seven invariants $I_1,I_2,I_4,I_5,I_6,I_7,I_8$ defined by [28]

$$I_1=\mathrm{tr}C,\quad I_2=\tfrac{1}{2}(I_1^2-\mathrm{tr}C^2),\quad I_4=\boldsymbol{M}\cdot C\boldsymbol{M},\quad I_5=\boldsymbol{M}\cdot C^2\boldsymbol{M},$$

$$I_6=\boldsymbol{M}'\cdot C\boldsymbol{M}',\quad I_7=\boldsymbol{M}'\cdot C^2\boldsymbol{M}',\quad I_8=\boldsymbol{M}\cdot C\boldsymbol{M}', \tag{2.1}$$

where C is the right Cauchy-Green strain tensor, and $\boldsymbol{M}$ and $\boldsymbol{M}'$ are the directions of the two families of fibers in the reference configuration. The Cauchy stress tensor is then given by

$$\begin{aligned}\sigma=&-pI+2\Psi_1B+2\Psi_2(I_1B-B^2)+2\Psi_4\boldsymbol{m}\otimes\boldsymbol{m}\\&+2\Psi_5(\boldsymbol{m}\otimes B\boldsymbol{m}+B\boldsymbol{m}\otimes\boldsymbol{m})+2\Psi_6\boldsymbol{m}'\otimes\boldsymbol{m}'\\&+2\Psi_7(\boldsymbol{m}'\otimes B\boldsymbol{m}'+B\boldsymbol{m}'\otimes\boldsymbol{m}')+\Psi_8(\boldsymbol{m}\otimes\boldsymbol{m}'+\boldsymbol{m}'\otimes\boldsymbol{m}),\end{aligned} \tag{2.2}$$

where p is the pressure associated with the constraint of incompressibility, B is the left Cauchy-Green strain tensor, $\Psi_i=\partial\Psi/\partial I_i$ $(i=1,2,\ldots,8)$, and $\boldsymbol{m}=F\boldsymbol{M},\boldsymbol{m}'=F\boldsymbol{M}'$ with F being the deformation gradient.

We shall choose a common cylindrical polar coordinate system, with basis vectors $\boldsymbol{e}_r,\boldsymbol{e}_\theta,\boldsymbol{e}_z$, to describe vectors and tensors in both the current and reference configurations. Thus, we may write

$$\boldsymbol{M}=\cos\phi\boldsymbol{e}_\theta+\sin\phi\boldsymbol{e}_z,\quad \boldsymbol{M}'=\cos\phi\boldsymbol{e}_\theta-\sin\phi\boldsymbol{e}_z, \tag{2.3}$$

where ϕ is the constant angle between the collagen fibers and the circumferential direction.

We consider the problem of axisymmetric inflation of a straight artery that has constant wall thickness H and uniform mid-plane radius R before inflation. Thus, in general, the axisymmetric deformed configuration may be described by

$$r=r(Z),\quad z=z(Z), \tag{2.4}$$

where Z and z are the axial coordinates of a representative material particle before and after inflation, respectively, and r is the mid-plane radius after inflation.

Since the deformation is axially symmetric, the principal directions of stretch coincide with the lines of latitude, the meridian and the normal to the deformed surface. Denoting the unit vectors in these principal directions by $\boldsymbol{e}_1,\boldsymbol{e}_2,\boldsymbol{e}_3$, respectively, we have

$$\boldsymbol{e}_1=\boldsymbol{e}_\theta,\quad \boldsymbol{e}_2=\cos\gamma\boldsymbol{e}_z+\sin\gamma\boldsymbol{e}_r,\quad \boldsymbol{e}_3=-\sin\gamma\boldsymbol{e}_z+\cos\gamma\boldsymbol{e}_r, \tag{2.5}$$

where γ is the angle between the meridian and the z-direction; see Fig. 1. The associated principal stretches are given by

$$\lambda_1=\frac{r}{R},\quad \lambda_2=\sqrt{r'^2+z'^2},\quad \lambda_3=\frac{h}{H}, \tag{2.6}$$

where h denotes the deformed wall thickness and the primes indicate differentiation with respect to Z. In the following analysis, we use R as the unit of length, which is equivalent to setting $R=1$.

The deformation gradient F may be written as

$$F=\lambda_1\boldsymbol{e}_1\otimes\boldsymbol{e}_\theta+\lambda_2\boldsymbol{e}_2\otimes\boldsymbol{e}_z+\lambda_3\boldsymbol{e}_3\otimes\boldsymbol{e}_r. \tag{2.7}$$

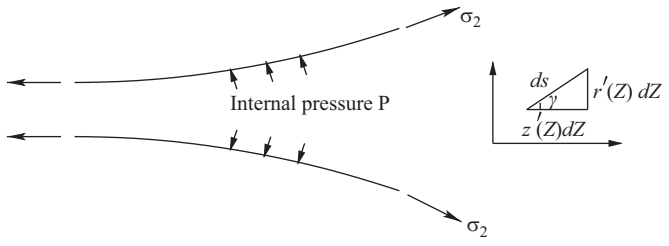

Fig. 1. Axisymmetric deformation of an artery.