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Modeling rupture of growing aneurysms

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ABSTRACT

Growth and rupture of aneurysms are driven by micro-structural alterations of the arterial wall yet precise mechanisms underlying the process remain to be uncovered. In the present work we examine a scenario when the aneurysm evolution is dominated by turnover of collagen fibers. In the latter case it is natural to hypothesize that rupture of individual fibers (or their bonds) causes the overall aneurysm rupture. We examine this hypothesis in computer simulations of growing aneurysms in which constitutive equations describe both collagen evolution and failure. Failure is enforced in constitutive equations by limiting strain energy that can be accumulated in a fiber. Within the proposed theoretical framework we find a range of parameters that lead to the aneurysm rupture. We conclude in a qualitative agreement with clinical observations that some aneurysms will rupture while others will not.

1. Introduction

Aneurysms are abnormal dilatations of vessels in the vascular system, and they exist in two major forms: fusiform and saccular. Fusiform aneurysms are found in the human abdominal aorta. Saccular aneurysms are found in cerebral blood vessels. The Brain Aneurysm Foundation (http://www.bafound.org/) reports that 2 in 100 people in US have an unruptured brain aneurysm and the annual rate of rupture is about 8–10 per 100,000 people. There is a brain aneurysm rupture every 18 minutes. Ruptured brain aneurysms are fatal in about 40% of cases. Of those who survive, about 66% suffer some permanent neurological deficit. Similarly, abdominal aortic aneurysm (AAA) is found in ~2% of the elderly population, with ~150,000 new cases diagnosed each year, and the occurrence is increasing (Bengtsson et al., 1996; Ouriel et al., 1992). In many cases AAA gradually expands until rupture causing a mortality rate of 90%. The AAA rupture is considered the 13th most common cause of death in US (Patel et al., 1995).

Medical doctors consider a surgery option for enlarging AAA, for example, when its maximum diameter reaches 5.5 cm or/and expansion rate is greater than 1 cm per year. This simple geometrical criterion may possibly underestimate the risks of rupture of small aneurysms as well as overestimate the risks of rupture of large aneurysms. Biomechanical approaches to modeling aneurysm failure are desired.

Watton et al. (2004) pioneered mathematical modeling of enlarging aneurysms. They described evolution of various arterial constituents including collagen and elastin. An interesting feature of their work is an explicit notion of the deformation corresponding to fiber recruitment. Most other fiber deformation models do not account for fiber recruitment explicitly yet introduce the phenomenon implicitly with the help of U-type (with significant stiffening) stress-strain curves. Baek et al. (2006) made another important step in modeling aneurysm growth by introducing a very convenient description of evolving strain energy density function – see formula (1) below. Building on the approaches mentioned above Kroon and Holzapfel (2007) developed aneurysm model which was attractive due to its theoretical and computational simplicity. The described works influenced further studies in mathematical modeling of aneurysm growth: Kroon and Holzapfel (2008; 2009); Chatziprodromou et al. (2007); Watton et al. (2009); Figueroa et al. (2009); Watton and Hill (2009); Schmid et al. (2010); Watton et al. (2011); and Martufi and Gasser (2012) to list a few. Though biomechanical features of intracranial and abdominal aortic aneurysms have differences (Humphrey and Taylor, 2008) the mathematical grounds of the G&R description can be common in both cases. Most mentioned theories consider turnover of collagen fibers as the main scenario of the aneurysm evolution.

Despite the success in describing growth and remodeling all mentioned theories were short of a failure description that should be a natural component of the theory. Volokh and Vorp (2008) proposed a new paradigm of Growth–Remodeling–Failure (G&R&F) by enforcing failure in a description of growth and remodeling. A failure description was enforced with the help of the *energy limiter* constant which provided a saturation value for the strain energy function (Volokh, 2011; 2013). The new constant controlled material failure and it could be interpreted as an

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average energy of molecular bonds from the microstructural standpoint. It is especially noteworthy that the approach of energy limiters allowed considering *strength* independently of *stiffness*. The latter separation is critical for the aneurysm modeling where *stiffening* can be accompanied by the *loss of strength*.[1]

The mentioned work by Volokh and Vorp (2008) used a purely phenomenological approach and was not guided by micro-structural considerations. Such considerations are taken into account in the present work in which we hypothesize that rupture of individual fibers (or their bonds) causes the aneurysm overall rupture. We examine this hypothesis in computer simulations of growing aneurysms in which constitutive equations describe both collagen evolution and failure. Failure is enforced in constitutive equations by limiting strain energy that can be accumulated in a fiber. Within the proposed theoretical framework we find a range of parameters that lead to aneurysm rupture. We conclude in a qualitative agreement with clinical observations that some aneurysms will rupture while others will not.

2. Methods

Most models of aneurysm growth and remodeling that appear in the works cited above, or the references therein, use fiber-based microstructural approaches. Any of these models can be enhanced with a failure description in the way it is done in the present paper. Following Humphrey and Rajagopal (2002), Baek et al. (2006), and, especially, Kroon and Holzapfel (2007) we assume that the aneurysm can be modeled as a membrane composed of collagen layers with the strain energy of the i^{th} layer prescribed in the form

$$\psi_i(t) = \int_{-\infty}^{t} g(t,t_{dp})\dot{m}_i(t_{dp}) f_i(t,t_{dp}) dt_{dp} \tag{1}$$

where $\dot{m}_i$ is the rate of the collagen fiber production; f_i is the strain energy of the deposited fiber; t_{dp} is the time of the fiber deposition; and the life cycle function $g(t,t_{dp})$ is defined by the fiber life time t_{lf} with the help of the Heaviside step functions H as follows

$$g(t,t_{dp}) = H(t-t_{dp}) - H(t-t_{dp}-t_{lf}) \tag{2}$$

In order to define constitutive laws for the rate of the fiber production and the fiber energy we have, first, to define kinematics of a fiber. We assume that $\mathbf{M}$ is a unit vector in the initial configuration at time $t=-\infty$ which defines direction of fiber deposition in the ith layer. Then, at time $t=t_{dp}$ a new fiber is deposited in direction

$$\mathbf{M}_{dp} = \mathbf{F}(t_{dp})\mathbf{M} \tag{3}$$

where $\mathbf{F}(t_{dp})$ is the deformation gradient mapping the initial configuration at time $t=-\infty$ to the configuration at time $t=t_{dp}$.

The deposited unit fiber $\mathbf{M}_{dp}/|\mathbf{M}_{dp}|$ is further mapped into[2]

$$\mathbf{m} = |\mathbf{M}_{dp}|^{-1}\mathbf{F}_{dp}\mathbf{M}_{dp} = |\mathbf{M}_{dp}|^{-1}\mathbf{F}_{dp}\mathbf{F}(t_{dp})\mathbf{M} = |\mathbf{M}_{dp}|^{-1}\mathbf{F}(t)\mathbf{M}, \tag{4}$$

where $\mathbf{F}_{dp} = \mathbf{F}(t)\mathbf{F}^{-1}(t_{dp})$ is the deformation gradient mapping material configuration at the time of the fiber deposition $t=t_{dp}$ to the current configuration at time t.

Besides kinematics we also prescribe a specific form of the fiber strain energy function in the i^{th} layer that enforces a failure description (Volokh 2011, 2013).

$$f_i(t,t_{dp}) = 0.1\,\Phi_i\{\Gamma[0.1,0] - \Gamma[0.1,(W_i(t,t_{dp})/\Phi_i)^{10}]\} \tag{5}$$

where $\Gamma[s,\ x] = \int_x^\infty t^{s-1}\exp(-t)\,dt$ is the upper incomplete gamma function; Φ_i is the energy limiter for fiber in the i^{th} layer; and W_i is the strain energy of intact (without failure) fiber in the ith layer.

We further specify constitutive equations as follows

$$W_i(t,t_{dp}) = \mu(\lambda_{pre}^2|\mathbf{m}|^2 - 1)^3 \tag{6}$$

$$\dot{m}_i(t_{dp}) = \beta\ |\mathbf{M}_{dp}|^{2\alpha} \tag{7}$$

where μ is a fiber stiffness parameter; λ_{pre} is a pre-stretch of the deposited fiber;β and α are the growth constants.

At this point the constitutive description is accomplished while a structural description is necessary. We restrict considerations by axisymmetric membranes. A membrane is in equilibrium when the virtual work of internal forces, $\delta\Pi_1$, is equal to the virtual work of external forces, $\delta\Pi_2$, or

$$\delta\Pi = \delta\Pi_1 - \delta\Pi_2 = 0 \tag{8}$$

The virtual work of the internal forces can be calculated by varying the total strain energy of the membrane

$$\delta\Pi_1 = \delta\int \psi\, dV \tag{9}$$

where $\psi = \sum_i \psi_i$ is the strain energy density per unit reference volume V of the membrane.

The virtual work of external forces is the virtual work of pressure, p,

$$-\delta\Pi_2 = -p\int_0^l 2\pi\, r\mathbf{n}\cdot\delta\mathbf{x}\, ds = 2\pi p\int_0^l r\left(\frac{dz}{ds}\delta r - \frac{dr}{ds}\delta z\right)ds \tag{10}$$

where

$$\mathbf{n} = \begin{pmatrix}\cos\alpha\\ 0\\ \sin\alpha\end{pmatrix} = \begin{pmatrix}-dz/ds\\ 0\\ dr/ds\end{pmatrix},\quad \delta\mathbf{x} = \begin{pmatrix}\delta r\\ 0\\ \delta z\end{pmatrix} \tag{11}$$

and s is the arc length of the membrane surface – see Fig. 1.

We note that it is possible to transform integral (10) over the current configuration to the integral over a reference configuration by introducing the reference arc length, S, in a way that the current arc length is a unique function of the referential arc length: $s(S)$. After such a transformation we have

$$-\delta\Pi_2 = 2\pi p\int_0^L r(z'\delta r - r'\delta z)dS \tag{12}$$

where primes designate derivatives with respect to the referential arc length and $l=s(L)$.

Remarkably, it is possible to introduce the pressure potential explicitly (Fried, 1982)

$$-\Pi_2 = \int_0^L \gamma(r,z')\,dS,\quad \gamma(r,z') = p\pi\, r^2 z' \tag{13}$$

Indeed, varying (13) we get (12)

$$-\delta\,\Pi_2 = \int_0^L\left(\frac{\partial\gamma}{\partial r}\delta r - \frac{\partial^2\gamma}{\partial S\partial z'}\delta z\right)dS = 2\pi p\int_0^L r(z'\delta r - r'\delta z)\,dS \tag{14}$$

Thus, equilibrium is provided by the stationary state of the total potential

$$\Pi = \Pi_1 - \Pi_2 = \int\psi\, dV + p\pi\int_0^L r^2 z'\, dS \tag{15}$$

This problem is conservative!

In the case of a membrane comprising n thin layers we can further simplify (15) as follows:

$$\Pi = \pi\int_0^L\left(2R\sum_{i=1}^{n} h_i\psi_i + pr^2z'\right)dS \tag{16}$$

where R is the referential or initial radial coordinate; h_i and ψ_i are the thickness and the strain energy of the ith layer accordingly.

We can specify equations written above by describing deformation in principal stretches

$$\mathbf{F} = \lambda_1\boldsymbol{\tau}\otimes\boldsymbol{\tau}_0 + \lambda_2\boldsymbol{\omega}\otimes\boldsymbol{\omega}_0 + \lambda_3\mathbf{n}\otimes\mathbf{n}_0 \tag{17}$$

where

$$\lambda_1 = s' = \sqrt{r'^2+z'^2},\quad \lambda_2 = \frac{2\pi r}{2\pi R} = \frac{r}{R},\quad \lambda_3 = \frac{1}{\lambda_1\lambda_2} \tag{18}$$

$$\boldsymbol{\tau} = \begin{pmatrix}\sin\alpha\\ 0\\ -\cos\alpha\end{pmatrix} = \begin{pmatrix}dr/ds\\ 0\\ dz/ds\end{pmatrix},$$

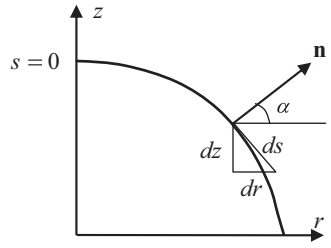

Fig. 1. Membrane of revolution.

[1] Remarkably, continuum damage mechanics theories usually describe failure through decrease of stiffness while the aneurysms failure is accompanied by increase of stiffness.

[2] See Remark 2 below.