



Short communication

# Thrombus rupture via cavitation

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## ABSTRACT

Aneurysm growth is accompanied by formation of intraluminal thrombus. The onset of thrombus rupture via unstable void growth is studied in the present note. The experimentally calibrated constitutive model of thrombus developed by Wang et al. (2001) is enhanced with a failure description and used for analysis of cavitation. It is found that unstable cavity growth can start at hydrostatic tension of 0.18 MPa which lies within the physiological range of stresses in the arterial wall.

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## 1. Introduction

Aneurysms are local dilatations in arteries that are prone to rupture. Often, AAA gradually expands until rupture causing high (90%) mortality rates. Medical doctors consider a surgery option for enlarging AAA when its maximum diameter reaches 5.5 cm or/and expansion rate is greater than 1 cm/year. These unsophisticated geometrical criteria may underestimate the risk of rupture of small aneurysms as well as overestimate the risk of rupture of the large ones. Biomechanical approaches to modeling aneurysm evolution have been developed and reviewed in Vorp (2007), Humphrey and Holzapfel (2012), and Pierce et al. (2015).

Despite a variety of biomechanical models of the aneurysm evolution the rupture *mechanism* is not clear. Ideally, the wall stress should increase with the increasing diameter and decreasing thickness of the arterial wall. The stress in the normal arterial wall is about 0.2 MPa (Humphrey et al., 2014) while its strength is about 1.2 MPa. Thus a six-time increase in stress can lead to rupture, assuming that the aneurysmal wall has strength similar to the healthy artery<sup>1</sup> (Vorp, 2007; Pierce et al., 2015). In reality, however, the aneurysm evolution is accompanied by formation and growth of intraluminal thrombus (ILT) which suppresses the stress increase (Vorp, 2007). Thus, the mentioned simple scenario might fail to reveal the process of mechanical rupture.

The fact that mechanical failure can occur before the material strength is reached can be explained by the existence of small defects that cause stress/strain concentration. For example, the aneurysm calcification (Inzoli et al., 1993; Basalyga et al., 2004; Marra et al., 2006; Speelman et al., 2007; Li et al., 2008; Maier et al., 2010; Buijs et al., 2013; O'Leary et al., 2015) would mean the appearance of rigid particles in the soft tissue. Such particles are

restraints that cause strain concentration from the standpoint of mechanics. Debonding of the tissue from the rigid particle or cavity growth in the vicinity of the particle is a possible mechanism of tissue failure. Such mechanisms are well known for materials varying from ductile metals to soft polymers (e.g. Benzerga and Leblond 2010).

In the recent study – Volokh (2015) – we examined cavitation instability in aneurysmal tissue based on the experimentally calibrated constitutive models. We found that the critical stress of cavitation instability could be significantly lower than the aneurysmal strength. However, we did not consider cavitation instability in ILT. The latter gap is filled in the present note. For the detailed methods of analysis the reader is referred to Volokh (2015) to which the present work is complementary.

## 2. Methods and results

We consider the experiments and ILT theoretical model reported in Wang et al. (2001) as the basis for the subsequent analysis. Moreover, we focus on the luminal region of ILT which exhibits the largest stiffness and strength among all ILT regions (abluminal, medial, and luminal). Wang et al. (2001) introduced and calibrated the following strain energy function for the luminal region of ILT:

$$W = c_1(I_2 - 3) + c_2(I_2 - 3)^2, \quad (1)$$

$$c_1 = 0.0337[\text{MPa}], \quad c_2 = 0.0347[\text{MPa}], \quad (2)$$

$$2I_2 = (\text{tr } \mathbf{B})^2 - \text{tr}(\mathbf{B}^2), \quad \mathbf{B} = \mathbf{F}\mathbf{F}^T, \quad \det \mathbf{F} = 1, \quad (3)$$

where  $\mathbf{F} = \partial \mathbf{y}(\mathbf{x})/\partial \mathbf{x}$  is the deformation gradient in which  $\mathbf{y}(\mathbf{x})$  is the current placement of a material point which occupied position  $\mathbf{x}$  in a reference configuration.<sup>2</sup>

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<sup>1</sup> Indeed, collagen fibers – the main load-bearing part of the arterial wall – are present in both healthy and diseased tissues.

<sup>2</sup> For the general background in nonlinear solid mechanics the reader is advised to consult Holzapfel (2000), for example.

In the case of uniaxial tension the constitutive law for Cauchy stress takes the form

$$\sigma = 2(\lambda - \lambda^{-2})W_2, \quad W_2 = \partial W / \partial I_2 = c_1 + 2c_2(I_2 - 3), \quad I_2 = 2\lambda + \lambda^{-2}, \quad (4)$$

where  $\lambda$  is the stretch in the tension direction.

The stress–stretch curve defined by (4) is presented in Fig. 1.

For the given constitutive model cavity growth curve is presented in Fig. 2 in which  $g$  is the remote hydrostatic tension and  $A$  and  $a$  are the cavity radii before and after deformation respectively. For the sake of brevity we completely omit the details of analysis that can be found in Volokh (2011) or Volokh (2015).

We emphasize that the graph is correct for any small cavity and it does not depend on the specific cavity size.

We note that the process of void growth is completely stable. The latter is a result of the use of the constitutive model of intact material presented by (1)–(3). Evidently, the Wang et al. (2001) material model cannot describe cavitation instability.

We modify the constitutive model of intact material by enhancing it with a failure description. We use a variant of the continuum description of bulk failure – softening hyperelasticity or elasticity with energy limiters (e.g., Volokh, 2013, 2014). Softening hyperelasticity is dramatically simpler in formulation than any existing approach for modeling material failure: its basic idea is to introduce an energy limiter in the expression for strain energy. Such limiter enforces saturation – the failure energy – in the strain energy function, which indicates the maximum amount of energy that can be stored and dissipated by an infinitesimal material volume during rupture. The limiter bounds stresses in the constitutive equations automatically.

Particularly, we introduce the strain energy function in the form

$$\psi = \frac{\phi}{10} \Gamma\left(\frac{1}{10}, 0\right) - \frac{\phi}{10} \Gamma\left(\frac{1}{10}, \frac{W^{10}}{\phi^{10}}\right), \quad (5)$$

where  $\Gamma(s, x) = \int_x^\infty t^{s-1} e^{-t} dt$  is the upper incomplete gamma function;  $W$  is the strain energy of intact, i.e. without failure, material presented by (1) and  $\phi$  is the energy limiter.

We calibrate the energy limiter based on the Wang et al. (2001) data on the strength  $\sigma_T = 0.53 \pm 0.07$  [MPa] in uniaxial tension experiments as follows:

$$\phi = 0.184 \pm 0.03 \text{ [MPa]}, \quad (6)$$

The stress–stretch curve for uniaxial tension of the material model with failure (6)–(7) is shown in Fig. 3. The limit point corresponds to the tensile strength and the onset of material failure.

The results of the analysis of cavity growth based on the constitutive model with the energy limiter are presented in Fig. 4.

The critical hydrostatic tension

$$g^c = 0.18 \pm 0.02 \text{ [MPa]}, \quad (7)$$

corresponds to the onset of the cavity yielding. This critical point indicates the cavitation instability which is defined as an event when the increase of the cavity size does not require further increase of the load.

We emphasize that the critical tension stress lies within the physiological range of stresses in the arterial wall.

### 3. Conclusions

We analyzed cavitation instability of intraluminal thrombus. For this purpose, we enhanced the experimentally calibrated

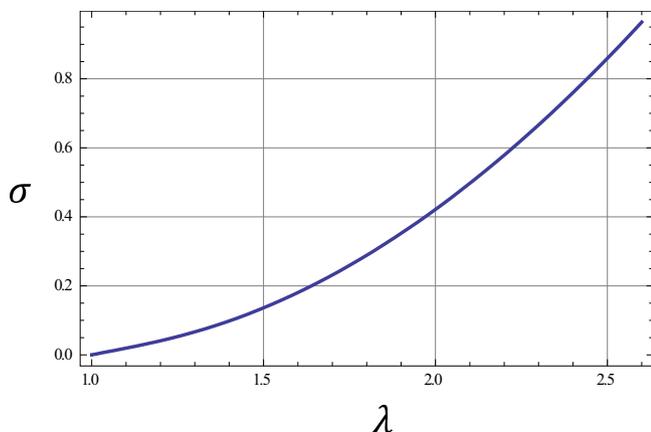


Fig. 1. Uniaxial tension: Cauchy stress [MPa] versus stretch for ILT material from Wang et al. (2001).

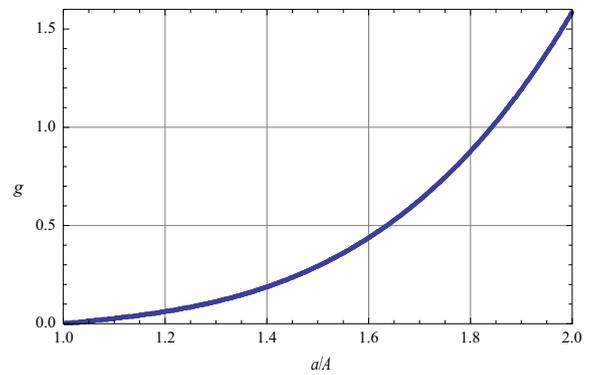


Fig. 2. Cavity hydrostatic tension [MPa] versus hoop stretch for thrombus material from Wang et al. (2001).

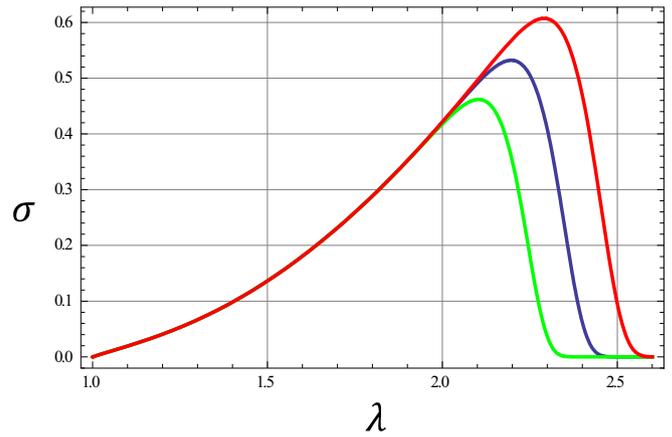


Fig. 3. Uniaxial tension: Cauchy stress [MPa] versus stretch for thrombus material with the varying strength.

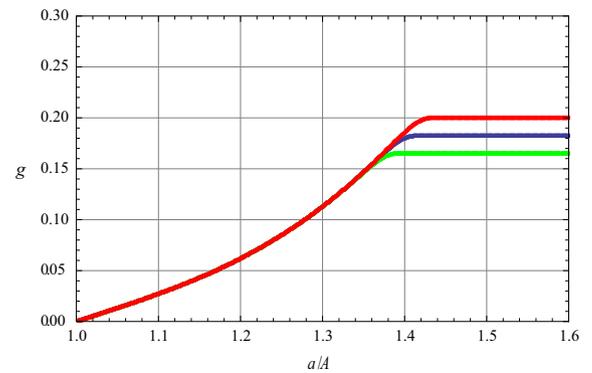


Fig. 4. Cavity hydrostatic tension [MPa] versus hoop stretch for thrombus material with the strength varying in accordance with Fig. 3.

constitutive model of Wang et al. (2001) with a failure description by introducing a limiter in the strain energy function. Such enhancement allowed us to describe the onset of the cavity yielding under remote hydrostatic tension. We found that the critical tension had the value of  $0.18 \pm 0.02$  [MPa] which correlated well with the stress in the arterial wall within the physiological range ( $\sim 0.2$  MPa).

Our finding suggests that cavitation instability can be a reason for the growth and coalescence of voids in the thrombus. The latter coalescence can lead to the formation of macroscopic cracks and ILT rupture. The thrombus rupture, in its turn, can lead to the initiation of the whole aneurysm rupture. For example, it might happen that the ruptured thrombus does not shield stresses anymore which

raise in the aneurysmal arterial wall. This stress rise might reach the aneurysmal strength directly or it might trigger a similar cavitation instability mechanism in the aneurysmal wall – Volokh (2015).

Cavity yields in hydrostatic tension. The latter state of deformation can occur near rigid inclusions. Thus, calcification, for example, can be a qualitative indicator of the danger of ILT rupture. More quantitative criteria need to be uncovered.

We note finally that the proposed failure mechanism is not necessarily unique and other factors, like fatigue, can be important. These factors may dominate or combine in the aneurysm rupture mechanism which remains to be revealed. The role of anisotropy should not be ignored either. Recently, Tong et al. (2011) reported anisotropic properties of an ILT luminal layer. A study of the role of anisotropy is of interest and should be considered in the future.

### Conflict of interests

None declared.

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