Arterial Seperation: A Cohesive Zone Modelling Approach

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Abstract
An aortic dissection is a serious and life threatening condition where a tear occurs in the intimal layer of the aorta. Cohesive zone models can be used to describe the delamination between two surfaces [9-10]. AD is fundamentally a biomechanics problem involving an initial material failure and subsequent unstable buckling between the layers of elastic laminae throughout the aorta extending from proximal to distal sections. A cohesive zone model was then implemented at the boundary between the arterial layers. Simulations predict that the greater the initial residual stress in the intimo-medial layer, the greater the subsequent cross-sectional area reduction of the true lumen. The contribution of residual stress to AD development is significant, reducing the cross-sectional area of the lumen by 17.4%-26.9% yet it is not the only factor driving its propagation.

1. Introduction
Aortic Dissection (AD) occurs due to intimal injury which causes an initial separation of the arterial layers. The separation is thought to occur most commonly between the intima and medial. This intima-medial tear is the pre-cursor for crack propagation, which is thought to occur between the lamellar elastic layers of the media [2,3]. The propagation of this crack/buckle commonly results in the development of pathological blood flow between the medial layers. This blood flow exerts an internal media pressure which not only causes further separation of the layers, but also expansion of this pathological conduit known as the “False lumen” [4]. This is often problematic as it results in a reduction in blood flow to the major arteries which stem from the aorta; the celiac, the superior mesenteric (SMA), the renals, the inferior mesenteric (IMA), the intercostal, and the spinal arteries. If these arteries are occluded/malperfused the outcome for the patient can be catastrophic (paraplegia, renal failure, stroke, etc.) [5].

The potential for separation of the arterial layers is highly influenced by the strength of the inter-lamellar bonds. If there is any portion of the media in which the morphology of elastin and collagen is pathologically altered, as seen in hypertension-induced obstruction of the vasa vasorum [6-8], the strength of the bridge fibers between those lamellar layers is also affected. Cohesive zone models can be used to describe the delamination between two surfaces [9-10]. AD is fundamentally a biomechanics problem involving an initial material failure and subsequent unstable buckling between the layers of elastic laminae throughout the aorta extending from proximal to distal sections.

To date no in silico models have been developed to simulate fracture propagation leading to AD. In this study a cohesive zone modelling approach is developed to simulate AD. A fundamental examination of the role of intraluminal septum thickness and residual stress in AD development is presented.

2. Model Development
An aortic ring geometry was created consisting of two anatomical layers; a Neo-Hookean adventitial-medial layer with an elevated stiffness (to simulate mild hypoxia), and a Neo-Hookean intimo-medial layer. The Neo-Hookean parameters were calibrated from uniaxial tensile tests of ovine aortic specimens. The interface between the layers was then divided into 2 sections; a portion of weakened aortic wall, and a portion of healthy aortic wall (AD is a local phenomenon; therefore, initiation is assumed to depend on some underlying local aortic weakness). The weakened wall was implemented through decreasing the strength of the interface between the layers. A cohesive zone model (McGarry et al. [10]) was then implemented at the boundary between the arterial layers. The normal and tangential interface tractions are defined in Eq.’s 1&2 below:

\[ T_n(\Delta_n, \Delta_t) = \sigma_{\text{max}} \times \exp(1) \left( \frac{\Delta_n}{\sigma_{\text{max}}} \right) \exp \left( -\frac{\Delta_n^2}{\sigma_{\text{max}}^2} \right) \]

\[ T_t(\Delta_n, \Delta_t) = \tau_{\text{max}} \times \exp(1) \left( \frac{\Delta_t}{\tau_{\text{max}}} \right) \exp \left( -\frac{\Delta_t^2}{\tau_{\text{max}}^2} \right) \]

Debonding occurs at the interface if the calculated traction \( T_n \) or \( T_t \) at a given increment exceeds the prescribed maximum allowable traction before separation. \( \sigma_{\text{max}} \) is the maximum traction in the normal direction without normal separation and \( \tau_{\text{max}} \) is the maximum traction in the tangential direction. Traction in a given direction is a function of \( \Delta_n \) and \( \Delta_t \) and the strength in the given traction direction. The model predicts identical behavior in mode I and mode II fracture if \( \sigma_{\text{max}} = \tau_{\text{max}} \).

A slight geometric asymmetry was introduced in the ring geometry to represent a bleb in the artery wall. A minor stress concentration occurs due to such a geometric imperfection. A residual stress was applied to the intima-medial layer of the artery.
3. Results

As shown in Figure 1, simulations predict that the greater the initial residual stress in the intimo-medial layer, the greater the subsequent cross-sectional area reduction of the true lumen. The thickness of the intraluminal septum as a percentage of the total aortic thickness also was also seen to influence the amount of true lumen loss.

![Figure 1: Relationship between normalized residual stress in artery wall (\(\sigma_{\text{residual}} / \tau_{\text{max}}\)) and the length of the false lumen (\(\Delta s / (D_o + t_o)\)) for a range of values of septum thickness (\(t_s\)). (\(t_o\)) is the thickness of the intact aortic wall.](image)

Figure 2 shows the predicted size of the true lumen following crack propagation in the artery wall. An in-vivo image of an AD is shown for comparison. Significantly higher levels of dissection are observed in-vivo than computed by our model, suggesting that residual stress is not the only driving force in the propagation of arterial dissection. However, the contribution of residual stress to AD development is significant, reducing the cross-sectional area of the lumen by 17.4%–26.9%.

4. Discussion

The clinical implications of the role the intraluminal septum thickness plays in the propagation of an intra-lamellar tear may be profound; should a patient present with an acute AD and has a thick intraluminal septum, this may indicate that the false lumen is likely to reduce the relative size of the true lumen due to the residual stress present.

New imaging modalities such as time-resolved flow MRI will allow for the quantification of arterial wall motion. The detection of localized regions of deformation adjacent to localized regions of elevated tissue stiffness may indicate a high risk of the initiation and propagation of AD.

![Figure 2: In-vivo image from a computed tomography scan of a dissected aorta (left). The true lumen is depicted by a “TL” in the image. Aortic dissection computed using a cohesive zone model is shown for comparison (right).](image)

8. References