**MODELLING THE GROWTH AND STRESS IN ABDOMINAL AORTIC ANEURYSMS**

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## INTRODUCTION

An abdominal aortic aneurysm is a localised progressive dilation of the abdominal aorta. Growth of the aneurysm is associated with a weakening of the wall and the possibility of rupture. Improved mathematical models of aneurysms may lead to a greater understanding of the pathogenesis of the disease and yield improved criteria for rupture.

Structurally, the key components of the arterial wall are elastin and collagen. The dilation of the aneurysm is accompanied by a degradation of elastin. Arteries remodel in response to deviations in stress and strain, so that it is necessary to address how the collagen will remodel as the wall dilates. A view held in the medical profession and supported by a cell mediated mechanism detailed in (Humphrey 1999), is that there is an equilibrium level of strain for the collagen fibres which remodelling will act to maintain.

## MATHEMATICAL MODEL

In its unloaded state, the collagen in the arterial wall bears no load and is crimped, so a recruitment parameter $r$ is introduced to define the factor by which the unstrained tissue must be stretched for the collagen to begin to bear load (Watton 2001). The degradation of the elastin is prescribed and the collagen can maintain an equilibrium strain by a remodelling of $r$ and/or a thickening of the collagen.

These features are incorporated into a three-dimensional membrane model that incorporates the helical structure of the collagen in the media and adventitia (Holzapfel 2000), and the localised structural properties of the collagen fibres. The collagen fibres are modelled as microscopic cylinders with a preferred direction, and are assumed to have mechanical characteristics independent of the configuration at which they are recruited. Before recruitment their contribution to the strain energy is assumed to be zero. The remodelling variables that describe the onset of recruitment, $r(x_1, x_2, t)$ and the density $n(x_1, x_2, t)$ of the fibres relative to the undeformed tissue are functions of the spatial coordinates and time. They are specified for the differing pitches of fibres throughout the plane of the membrane. The definitions of the remodelling parameters allow for the total mass of collagen remains constant as the aneurysm develops. It is assumed that the fibres remodel to maintain their strain at systole to a constant. The spatial and temporal degradation of elastin is prescribed and differential equations are proposed to simulate the remodelling of the fibres due to deviations in the local strain field. Remodelling rates for the elastin and collagen are estimated from (Chang 1994) and (Humprey 1999) respectively. A variational equation governs the ensuing deformations which is solved by a finite element method.

## RESULTS

Using a physiologically determined set of parameters to model the abdominal aorta and realistic remodelling rates for its constituents, the predicted dilations of an axisymmetric aneurysm are consistent with those observed in vivo.

Three-dimensional deformations are analysed for two cases: (a) by imposing a localized non-axisymmetric degradation of elastin and (b) by using a stiff-backed plate to simulate aneurysm development with spinal contact contact. The stress and strain fields can be predicted for use in rupture criteria.

The model has also been employed to consider the growth of abdominal aortic aneurysms under different imposed conditions. For example, it predicts the increase in the rate of dilation in hypertensive conditions and the retraction of the aneurysm that follows a stent bypass ope ration.

Of fundamental importance is the fact the remodelling parameters are structurally based and thus have direct physical interpretation. Indeed, they allow for a visual interpretation of the remodelled structure of collagen.

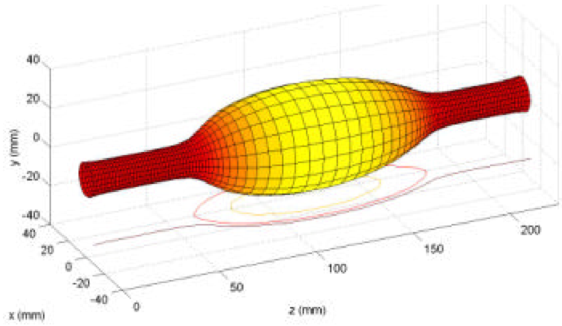


**Figure 1: An axisymmetric abdominal aortic aneurysm.**

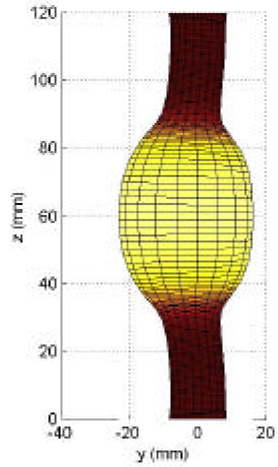


**Figure 2: Deformation of an abdominal aortic aneuysm subject to spinal contact.**

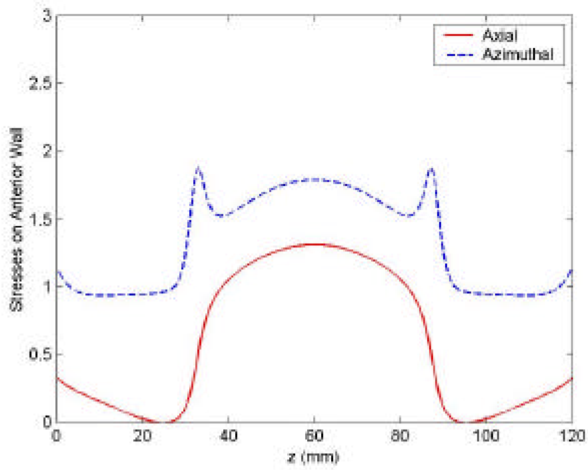


**Figure 3: Example of the stress in the wall of the aneurysm.**

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