PREDICTING THE GROWTH AND RUPTURE POTENTIAL OF ABDOMINAL AORTIC ANEURYSMS USING FINITE ELEMENT ANALYSIS

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Abstract: This research is concerned with the development of a technique to predict the pattern of growth and risk of rupture of individual patient's abdominal aortic aneurysm (AAA). This paper deals specifically with the development of a computerised growth prediction model based on the engineering technique of finite element analysis (FEA).
Currently the growth of an aneurysm is believed to be linked intrinsically with the material properties of the aneurysm wall. Also it is thought that the formation of an aneurysm is due mainly to the excessive action of certain matrix metalloprotease (MMP) on the elastin content of the aorta wall. This reduction in wall strength and elastic recoverability is also believed to be the factor sustaining continued growth of the AAA.
The model described here mimics the behaviour of the MMP's and selectively reduces the elastin content of the wall, then simulates the reaction of the collagen to compensate.
By this method, growth of an aneurysm has been simulated, with some very promising initial results. This paper discusses the results so far.

Introduction

In some people, as their age increases and the effects of too much alcohol and cigarettes take their toll, serious health problems frequently become an issue. Often the heart and arterial system are extremely susceptible to this aging process, possibly leading to an abdominal aortic aneurysm (AAA), which currently occurs in over 39,000 people in the UK [1]. An aneurysm is an inflammation of an artery or vein. In an abdominal aorta such a condition can lead to rupture of the aortic wall which is associated with 50% mortality before reaching hospital, and 40-50% mortality when in hospital undergoing emergency repair.

In some cases patients may be advised to opt for an elective repair, which is associated with a 3-6% mortality rate [2]. The decision on whether to perform an elective repair is currently based on a rule of thumb which says that if the maximum diameter of the AAA is greater than 5.5cm then the operation should go ahead [3]. However, although size is an important predictor of rupture, not all large AAA rupture, while around 10-24% of small ones do [4]. Currently no reliable criterion exists to predict risk of rupture on an individual patient basis.

Therefore this research is concerned with the development of a technique to predict the patient specific rupture potential of AAA. This paper focuses on part of that work which is concerned with the development of a reliable computer model for the conception and further growth of an aneurysm.

The aorta is not comprised of a homogeneous material. The wall consists of different components arranged in ascending layers through the wall. This composition gives the wall strength, to resist the systolic blood pressure, and elasticity, which allows expansion and contraction billions of times over one's life, without becoming brittle or rupturing. The general cross-sectional composition of the aorta is shown in figure 1, and is the basis of the modelling which comes later.

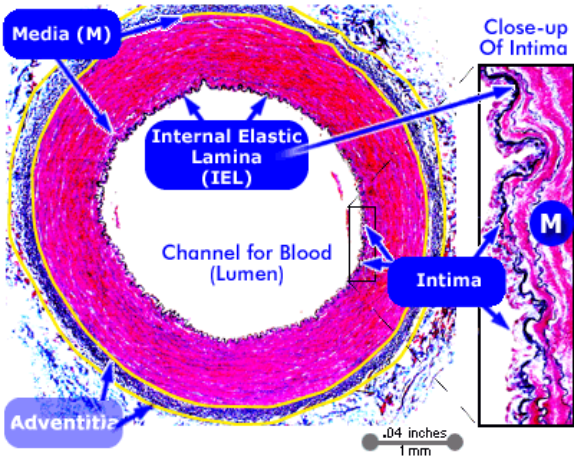

Figure 1: Aortic cross-sectional composition [5].

The wall is made up of three layers, the *tunica intima, tunica media and tunica adventitia*. The intima

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is a one cell thick layer of axially orientated endothelial cells, and an underlying basal membrane. It is believed that the intimal layer does not significantly add to the mechanical properties of the aortic wall. Thus in many studies where constitutive equations are developed for the aorta, there is no component attributed to the intimal response. [6,7,8]

The second layer is the *tunica media*, which consists mainly of smooth muscle embedded in a plexus of collagen and elastin within a ground substance gel matrix. Generally in elastic arteries the fibres of collagen in the media are orientated helically and arranged in concentric layers separated from each other and the intima and adventitia by fenestrated sheets of elastin. It is believed that due to the elastin content, the media is the part of the aorta that gives the bulk of the elastic recovery during loading.

The third layer is the *tunica adventitia* which consists of collagen fibres within admixed elastin. The adventitia is less stiff than the media in load free situations due to the tortuousness of the collagen fibres, but at higher expansions the collagen fibres straighten and make the adventitia into a stiff jacket which protects the artery from over stretching. [9]

The wall contains a significant amount of smooth muscle which it might be expected would affect the properties of the wall. However, it is reported in [10] that smooth muscle necrosis of the aorta in rats over a two month period had no significant effect on the wall mechanics. Therefore it appears that passive wall mechanics, not smooth muscle viability, is the primary determinate of wall stiffness [11], and so smooth muscles are not included in this present modelling work.

It is believed that aneurysms form in part because of damage to, or defects in, intra-luminal elastin and collagen [12, 13], where this damage is caused by an increase in proeolytic activity. There are a number of animal studies which have shown that elastase-induced degradation of intra-luminal elastin results in dilation of a previously normal wall [14, 15, 16]. Therefore the phenomena of aneurismal initiation can be explained by an initial increase in matrix metalloprotease (MMP) activity, leading to reduced elastin content of the wall, and initial expansion. Fortunately the collagen is strong enough to resist the extra stress and remodels to compensate for the new content of elastin in the wall thus limiting the expansion. The collagen remodels first because it can regenerate in minutes, while the elastin takes much longer, with a half life of years. The result is that the initial expansion of the wall due to the reduction of elastin is maintained permanently, and thus the AAA is formed.

As the collagen remodels in response to the expansion, the amount of collagen in the wall increases. Thus when the cycle of expansion and remodelling is repeated, the percentage of elastin content gradually reduces, and the collagen continues to compensate so that the overall percentage composition of collagen in the wall increases. This explanation is supported in [17] which reports that smooth muscle and elastin content decreased by around 90% in AAA, where as collagen and gel matrix content increased by 77%.

The ultimate end of these phases of growth, which in fusiform aneurysms typically occurs at a rate of 0.4cm per year [18,19], is rupture. This is not unexpected, since thin cylinder theory predicts that stress in the aneurysm wall will be proportional to radius. Thus:

$$\sigma_C = (P \times r)/t \qquad \sigma_L = (P \times r)/2t \qquad (1)$$

σ_C is the circumferential stress.
σ_L is the longitudinal stress.
P is the internal pressure of the cylinder.
r is the radius of the cylinder.
t is the wall thickness.

Thus at a fixed internal pressure, the wall stress will increase as the radius of the cylinder or aneurysm increases, so that eventually the strength of the wall material will be exceeded and rupture will occur. However, clinical experience shows that some small aneurysms rupture while other larger ones do not. Therefore predicting rupture based on radius alone is unreliable.

Method

This computer model is an attempt to recreate the above process in a way which is both realistic and repeatable. Therefore there are a number of aspects of the wall mechanics which must incorporated in the model. Firstly, although arteries are not truly incompressible, they appear to be nearly incompressible under physiologic loading. Consequently, incompressibility of the arterial wall is assumed in this research [11, 20]. Secondly, although previous research has assumed the aorta wall to be isotropic [21], the helical arrangement of the collagen fibres in the wall means that it behaves like an engineering composite [9] with orthotropic properties. Therefore, in this model the aortic wall is generated as a square lattice of fibres embedded in a matrix of elastin. This allows different circumferential and longitudinal fibre properties to be defined, and hence the effective orthotropy of the helical collagen fibres to be represented. In bovine specimens the helix angle is 29° to 62°, depending on the layer [22].

The elastin and collagen combination demonstrates a special behaviour in which the initial strain of the aorta wall is controlled by the elastin (figure 2). Because of the tortuous nature of the collagen fibres, a significant strain must be reached before they become taut and start to carry any of the load [23]. When they do become active, their significantly higher stiffness results in much stiffer wall behaviour.

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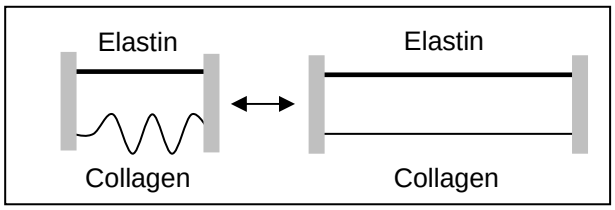

Figure 2: Collagen and elastin fibres under no tension and tension.

This fibre behaviour is implemented in the model by the use of link elements. These act like tension-only cables with a fixed positive offset and zero compressive stiffness. The behaviour of the elastin matrix and these link elements when stressed in tension is illustrated in figure 3.

When the elastin is depleted, the initial stiffness of the wall is reduced, and the collagen experiences a higher strain than it normally would do (figure 4). This additional strain leads to remodelling of the collagen and results in permanent deformation and expansion of the aneurysm (figure 5).

This analysis was undertaken using the ANSYS finite element analysis system. The elastin matrix was modelled with quadrilateral shell elements (ANSYS type shell1181). The collagen was represented by one dimensional link elements (ANSYS type link10) attaching to the corner nodes of the shell elements. These fibres were aligned longitudinally and circumferentially with both directions having a unique strain offset.

The different material properties used in the model are shown in figure 6. The dark blue line shows the elastic response that was assumed for *normal* elastin, while the light blue line shows the elastic response of the elastin after MMP degradation. This simulates a 93% reduction in elastin content causing the same reduction in elastic stiffness, where 93% is reported to be the maximum amount of elastin lost in a fully developed aneurysm [17].

In the model the initial elastin reduction occurred at a user-defined location and continued here for the duration of the simulation. This was to model the continued activity of the MMP's at this location throughout the expansion of the AAA. It is assumed that the MMP activity will spread, thus from this initial position the aneurysm will grow to encompass more of the aorta. To mimic this behaviour the model used the localised elevated stresses around the weakened site to drive the weakening of a larger proportion of the aorta.

In the simulation, the collagen was assumed to remodel to maintain the excess strain and thus the aorta expanded. Excess strains were those above the normal physiological conditions. These conditions are shown in figure 6; (green and orange line which show the maximum stress on the aortic wall at 120mmHg BP and for an aortic radius of 12.5 mm).

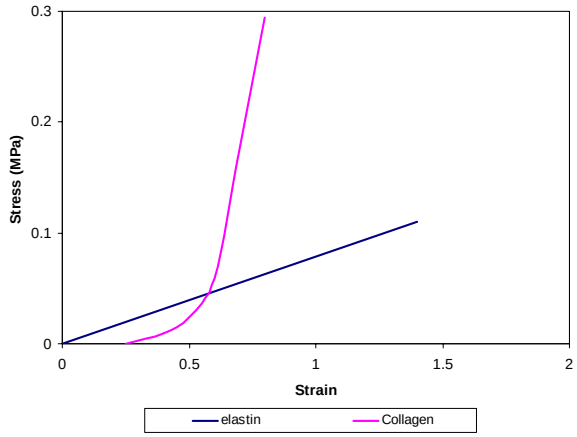

Figure 3: The normal two phase collagen and elastin response to tensional stress.

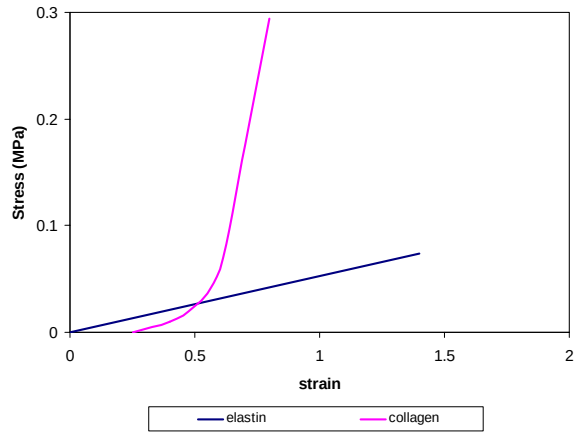

Figure 4: Depleted elastin and collagen under tensional stress.

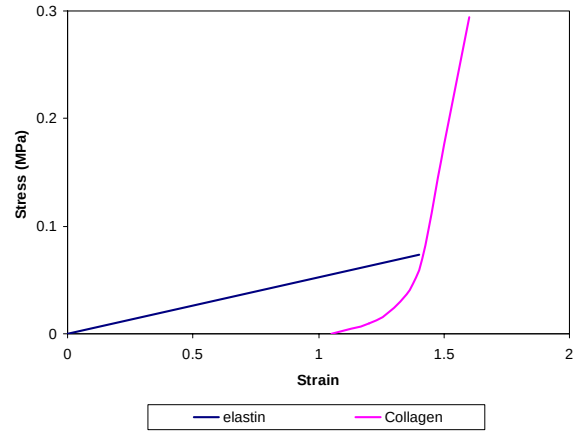

Figure 5: The new collagen and elastin wall characteristics with an increased collagen offset leading to the aneurismal expansion being maintained.

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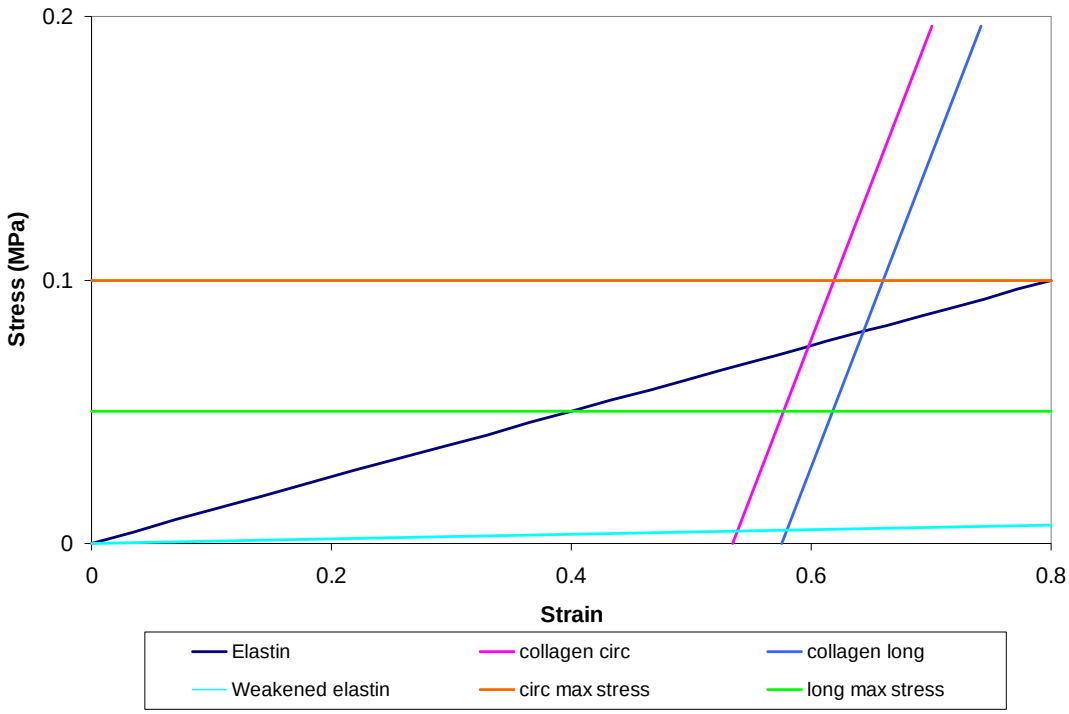

Figure 6: The material properties of the elastin and collagen in the computer model

Figures 7 and 8 show the collagen lattice and elastin matrix respectively. Under the conditions detailed above and with constraints applied to each end of the model (to mimic the tethering at the renal arteries and the aortic bifurcation), the model was pressurised and run in a stepwise fashion to approximate the growth of an aneurysm.

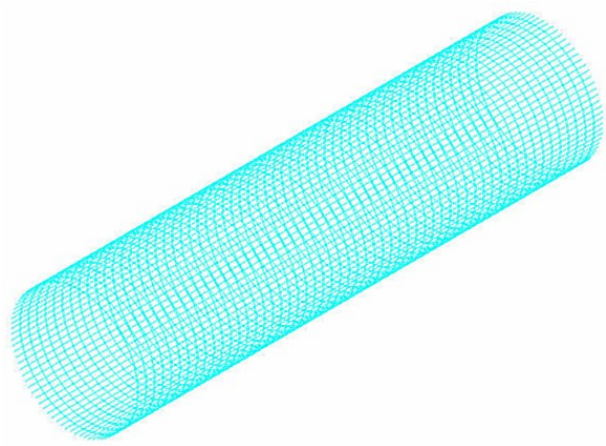

Figure 7: The collagen mesh in the computer model.

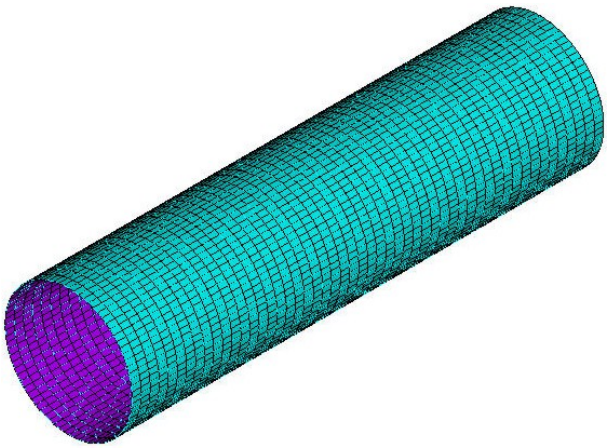

Figure 8: The elastin matrix in the computer model.

Results and discussion

Sample results are displayed in figures 9 and 10. Figure 9 shows the growth of a symmetric aneurysm, created by selecting a band around the circumference of the aorta for initial weakening. Figure 10 shows the expansion of an asymmetric aneurysm, initiated by weakening a portion on one side of the aorta.

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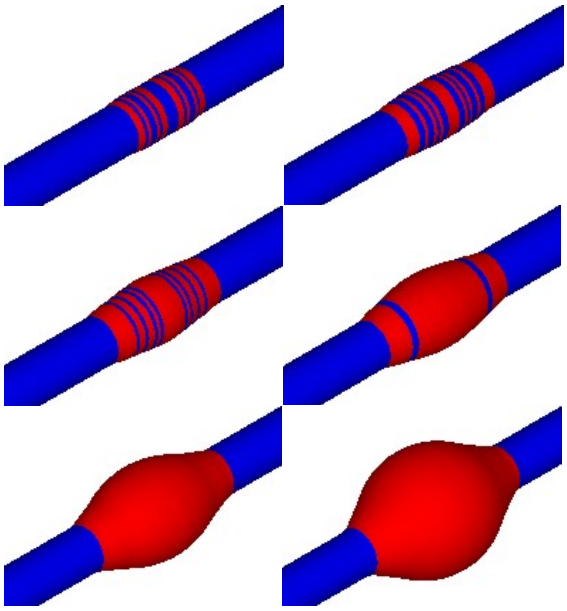

Figure 9: Results of initial weakening over a band of aorta.

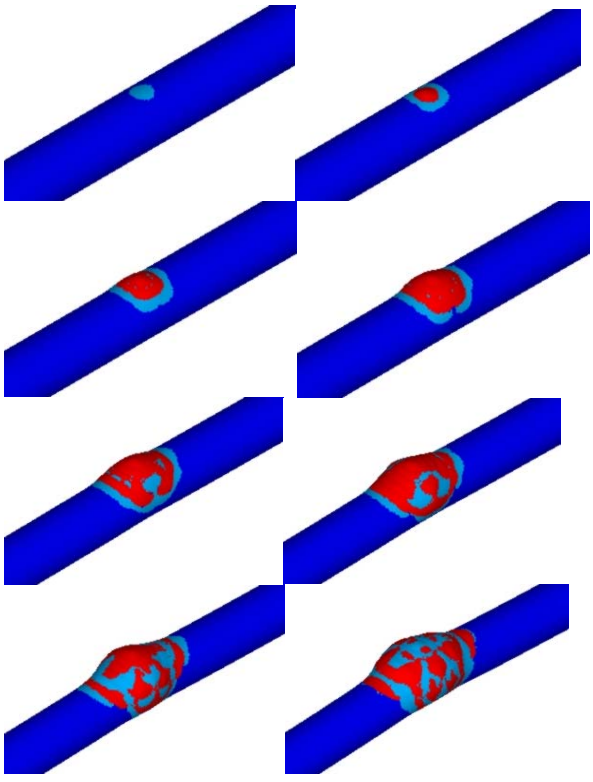

Figure 10: Results of initial weakening over a selected portion on one side of the aorta

The areas of red are the weakened materials and following the colour changes it can be seen how the model recruits more of the aortas surface area into the aneurysm. However, there are flaws in these results; the main area of concern is that the growth occurs in large steps. This may be due in part to the fact that the model does not take account of the wall thickening which has been observed in-vivo. Additionally there are some problems with the point of rupture, mainly attributed to anomalies in the rebuilding of nodes, after each iteration. It is hoped that with further development these problems can be overcome.

Conclusion

The initial model and results are simplistic but promising. It will be interesting to observe the different growth patterns and how the models cope when applied to real aneurysm geometries in an early state of development.

In the future, other body parts such as the spine will be included in the simulation to make the growth more realistic. In order to validate the growth prediction, comparisons will be made with consecutive CT patient scans.

This research has many implications for patients and health service funding. The research is aimed primarily at improving the longevity and quality of life of the elderly population by preventing them from experiencing a ruptured aneurysm or undergoing an elected but still very major surgical operation. This modelling procedure could eventually provide a method to estimate how a patient's aneurysm will grow and if it is at high risk of rupturing in the near future. Thus allowing a more informed decision making process for the surgeons who must decide whether an elective repair should go ahead. It is further hoped that through this research we can gain a better understanding of the pattern of the disease and why some aneurysms rupture at a smaller size than others.

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