Stress Analysis of the Diseased Arterial Cross-section

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ABSTRACT

There are a number of studies addressing the relationship between blood flow and the initiation and progression of atherosclerotic plaque. However, once a plaque develops, the central question becomes what hemodynamic, mechanical or other factors determine whether or not the plaque remains stable? A closely related question concerns the response of the plaque to mechanical manipulation as, for example, by balloon angioplasty.

As a pilot study, we have constructed a finite element model simulating the diseased arterial cross-section in plane stress. The model was used to determine the effects of geometric and mechanical property variables on stresses which may influence the stability of the plaque, e.g., the stresses at the interface between the inner plaque and the outer plaque/media. Our results suggest that differences in stiffness within the plaque, the size of the plaque and the relative size of the lumen significantly influence the magnitudes and distribution of stresses in the cross-section. The implications of these results both to the stability of the plaque and to the success of balloon angioplasty are discussed.

INTRODUCTION

Studies of hemodynamics have been successful in identifying the mechanical factors important to the initiation and development of atherosclerotic plaque (1). Although many questions remain to be answered, there is now good information relating sites of low and oscillatory shear stress to the distribution of intimal thickening and plaque at clinically important sites (2,3,4). Once the plaque develops, the artery wall may compensate for hemodynamic changes by dilating or otherwise changing its shape in order to maintain a lumen size consistent with adequate blood flow (4,5,6). Obstruction can occur when the artery cannot adapt as quickly as is necessary in response to plaque growth or when, for reasons not well understood, the plaque disrupts. Plaque disruption and degeneration lead to complications such as stroke and heart attack.

The stresses developed in the atherosclerotic plaque in response to the mechanical loading are influenced by the fact that, as the plaque evolves, it exhibits a complex microstructure and geometry. For example, plaques vary in consistency from soft and gelatin-like to calcific and stiff. Our working hypothesis is that the stresses in the plaque and artery wall are influenced by the hemodynamic forces engendered by pulsatile flow and may influence plaque stability. Preparatory to experimental work in the area, we seek to identify which geometric, mechanical, compositional or other features of atherosclerotic plaque have important effects on the distribution of stress in the cross-section of arteries.

MODEL

The arterial wall is non-homogeneous, at best orthotropic and exhibits both geometric and material nonlinearities (7). The added geometric complexity of modelling the diseased wall naturally leads to the finite element method of analysis (8). Although the problem considered here could have been attacked using finite differences, our long term experimental interests lead us to choose a finite element based approach.

The model studied, shown in Figure (1), consists of two non-concentric circles with fixed offset, the external one of radius R and the lumen of radius r. Important features of the model are stratification in the region of the plaque and the eccentric nature of the plaque. The interface between the inner plaque and the outer plaque/media was generated by fixing three points at (r,0), (-r,0) and (0,-[0.1 + d]) and passing an arc between them. Although this model represents a somewhat simplified view of plaque development, it is capable of simulating the potentially important effects of variable lumen radius (r), plaque size (d) and changes in the relative stiffness between the inner plaque and the outer plaque/media.
For simplicity, the arterial cross-section is assumed linear elastic and isotropic but with different moduli $E_p$ for the inner plaque and $E_m$ for the outer plaque/media. Poisson’s ratio is taken to be 0.49, consistent with the usual assumption that the arterial wall is incompressible or nearly so. The applied loading was taken as the mean blood pressure: 100 mmHg.

ANSYS (Swanson Analysis Systems) was used to implement a plane stress solution using six-node triangular elements. These elements are well suited for modeling complex geometry. The element has nodes at the vertices of the triangle and at mid-side and is formulated using quadratic displacement functions (9).

Figure (2) shows a representative mesh generation taking the symmetry of the model into account. Convergence studies indicated that roughly five hundred elements were sufficient.

**PARAMETER STUDY**

The number of parameters in the model was reduced by limiting the study to investigating the effects of inner plaque size, lumen radius and the relative stiffness between the inner plaque and the outer plaque/media. Hence the external radius $R$ was fixed at 0.5 cm. The geometric parameters $r$ (lumen size) and $d$ (inner plaque size) studied were $r = 0.1, 0.2$ and 0.3 cm and $d = 0.06, 0.12$ and 0.18 cm.

The mechanical properties of the plaque are unknown though there is some published experimental work on plaque tensile strength (10). The Young’s modulus of elastin is about $10^8$ Pa while that of collagen is about $10^9$ Pa (7). In our parameter study, we chose the modulus of the outer plaque/media $E_m = 10^7$ Pa and varied the modulus of the inner plaque ($E_p$) over the range $10^3 < E_p < 10^{11}$ Pa. For relatively large differences in modulus, the stresses at the interface between the two materials can be discontinuous. Hence we used stress averaging at the interface (8). Finite elements specifically formulated to account for material property gradients represent an alternative approach (11).

**RESULTS**

Representative results are depicted in Figures (3) - (5). Figure (3) illustrates the effect of the modulus ratio $E_p/E_m$ on the distribution of maximum principal stress. Shown in the Figure are lines of constant maximum principal stress for $E_p/E_m = 10^4$, $1, 10^4$ with $r = 0.3$ cm and $d = 0.06$ cm. Note that the location of the maximum principal stress shifts from the inner plaque interface (Figure 3-A) to the intima away from the plaque (Figure 3-C) as the stiffness of the inner plaque relative to the outer plaque/media changes from "hard" to "soft". Contour plots of the maximum shear stress (not shown) indicate that the effect of relative plaque stiffness on the distribution of maximum shear stress is similar to that described for maximum normal stress.

Figure (4) shows the effect of inner plaque size on the shear stress at the interface between inner plaque and outer plaque/media in the direction tangent to the interface. Shown is the shear stress at $\theta = 45^\circ$ for ratios of $E_p/E_m = 10^4, 1, 10^4$. Note that this stress increases with plaque size for relatively less stiff inner plaques but is not much affected by plaque size for relatively stiff inner plaques.

Figure (5) depicts the effect of lumen radius $r$ on the stress normal to the interface between inner plaque and outer plaque/media. Shown is the normal stress at $\theta = 45^\circ$ for ratios of $E_p/E_m = 10^4, 1, 10^4$. Note the significant effect of the relative stiffness between inner plaque and outer plaque/media.

**DISCUSSION**

Although it is too early to draw firm conclusions from this study it is interesting to introduce representative histological slides prepared from diseased human arterial cross-sections. Figure (6A) shows a typical plaque configuration while Figure (6B) shows an identical plaque after being subjected to rapid balloon dilatation (12). Note that the plaque has separated from the media and a narrow layer of underlying intimal thickening at the interface between the plaque and plaque/media, a region of high stress, at least for relatively stiff plaques.

Figure (7) shows a plaque, subject to balloon dilatation with the media torn (arrow). Our model predicts greater stresses away from the inner plaque - outer plaque/media interface for inner plaques which are relatively less stiff.

**CONCLUSION**

Stress analysis of the diseased arterial cross-section when coupled with relevant experiments and histological observations could shed new light on the mechanical factors related to plaque stability.

**REFERENCES**


**Figure (1)** Geometric model of a diseased arterial cross-section.

**Figure (2)** A representative discretization of the model using triangular elements is shown. The problem is symmetric.

**Figure (3)** The effect of changes in the relative stiffness of the inner plaque on the distribution of normal stress in the cross section is shown for $r = 0.3$ cm, $d = 0.06$ cm. (A) $E_p/E_m = 10^4$ (B) $E_p/E_m = 1$ (C) $E_p/E_m = 10^4$. Legend: $A - E = 1 \times 10^6$ Pa, $F = 60 \times 10^5$ Pa.

**Figure (4)** Shear stress tangent to the inner plaque - outer plaque/media interface at $\theta = 45^\circ$ vs relative stiffness $E_p/E_m$ for various choices of plaque size $d$. 
Figure (5) Normal stress perpendicular to the inner plaque - outer plaque/media interface at $\theta = 45^\circ$ vs relative stiffness $E_p/E_m$ for various choices of lumen radius $r$.

Figure (6) Histological slides of plaques: (A) typical multilayered plaque of complex composition and (B) plaque identical to (A) but having been subjected to rapid balloon dilatation.

Figure (7) Large plaque disrupted by balloon dilatation with torn media but intact adventitia at arrow.