

Effect of intraluminal thrombus on abdominal aortic aneurysm wall stress

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Purpose: Abdominal aortic aneurysms (AAAs) rupture when the wall stress exceeds the strength of the vascular tissue. Intraluminal thrombus may absorb tension and reduce AAA wall stress. This study was performed to test the hypothesis that intraluminal thrombus can significantly reduce AAA wall stress.

Methods: AAA wall stresses were determined by axisymmetric finite element analysis. Model AAAs had external diameters ranging from 2.0 to 4.0 cm. Model parameters included: AAA length, 6 cm; wall thickness, 1.5 mm; Poisson's ratio, 0.49; Young's modulus, 1.0 MPa; and luminal pressure, 1.6×10^5 dyne/cm². Stresses were calculated for each model without thrombus, and then were recalculated with thrombus filling 10% of the AAA cavity. Calculations were repeated as thrombus size was increased in 10% increments and as thrombus elastic modulus increased from 0.01 MPa to 1.0 MPa. Maximum wall stresses were compared between models that had intraluminal thrombus and the unmodified models. Stress reduction greater than 25% was considered significant.

Results: The maximum stress reduction of 51% occurred when thrombus with elastic modulus of 1.0 MPa filled the entire AAA cavity. Stresses were reduced by only 25% as modulus decreased to 0.2 MPa. Similarly, decreasing thrombus size by 70% resulted in stress reduction of only 28%. Large AAAs experienced greater stress reduction than small AAAs (48% vs 11%).

Conclusion: Intraluminal thrombus can significantly reduce AAA wall stress. (*J Vasc Surg* 1997;26:602-8.)

When tensile stresses inside the walls of an abdominal aortic aneurysm (AAA) exceed the natural strength of the wall tissues, material failure occurs, tissues separate, and aneurysm rupture ensues. Thus wall stresses play a pivotal role in aneurysm failure. Understanding how these stresses are distributed and what factors influence stress distributions is critical in evaluating the potential for rupture. Intraluminal thrombus can absorb tensile stresses and potentially shield the underlying aneurysm wall. However, the extent of this potential protection has yet to be systematically examined.

Finite element analysis is a numerical technique

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capable of modeling stresses in complex physical structures.¹ Finite element analysis has been used in simple aneurysm models to examine wall stress distributions and to determine how biomechanical and geometric factors affect these stresses.²⁻⁴ These investigations have examined how factors such as diameter, wall thickness, shape, and material properties alter aneurysm stress distributions, and previous work by Inzoli et al.⁴ suggests that under some situations intraluminal thrombus may be important in reducing AAA wall stresses.

The purpose of this study was to examine how intraluminal thrombus affects AAA wall stresses. Using finite element analysis, this study systematically examines how thrombus size, shape, and material properties interact with the size, shape, and material properties of AAAs to alter wall stress distributions.

METHODS

Basic modeling considerations. Using computer simulations, we modeled aneurysms as axially symmetric structures of revolution with walls constructed of homogeneous and isotropic materials that exhibit linear elastic deformation. We also assumed that our idealized aneurysms were not subject

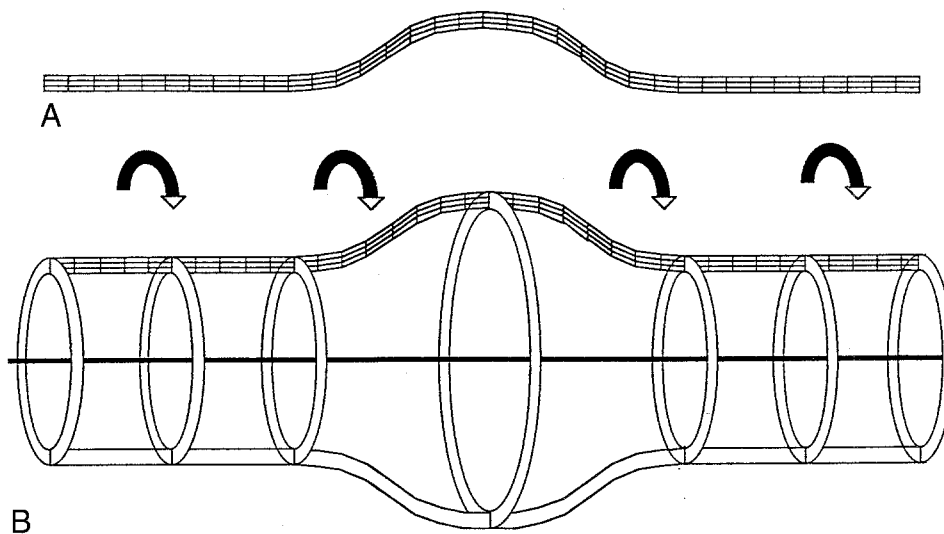


Fig. 1. **A,** Axisymmetric finite element grid representation of a small aneurysm. **B,** Rotation of the grid about the axis of symmetry reproduces the three-dimensional structure of the aneurysm.

to external forces, pressures, or extravascular tethering.

We geometrically represented aneurysms using three concentric layers of axisymmetric elements. Our use of axially symmetric elements implies that each grid element actually represents a toroidal section of aneurysm. We conducted mathematical convergence tests and determined that 138 elements would be needed to assure numerical accuracy. Fig. 1 illustrates a typical finite element aneurysm grid. This grid depicts a sagittal section through the anterior aneurysm wall. As illustrated, the three-dimensional aneurysm structure is obtained by rotating the grid about the axis of symmetry. The toroidal sections are generated by rotating individual elements about the symmetry axis.

We tethered both ends of a 6.0 cm aneurysmal aortic segment to 1.5 cm segments of normal vessel. We gave the normal aortic segments outer diameters of 2.0 cm^{5,6} and wall thicknesses of 1.5 mm.⁷ We used a sinusoidal curve to define the contour of the outer aneurysm wall. The resulting aneurysm had an outer diameter of 2.0 cm at the junction with the normal aorta and a maximal diameter of 4.0 cm over the dome. We prohibited motion along the axis of symmetry by tethering the distal ends of the normal aorta. We endowed our normal aortic segments and base aneurysms with the following material properties⁸⁻¹¹: elastic modulus, 1.0×10^7 dynes/cm²; shear modulus, 3.3×10^6 dynes/cm²; Poisson ratio, 0.49.

Using an approach similar to that used in the

aorta, we modeled intraluminal thrombi as axisymmetric structures using three concentric layers of quadrilateral elements. As in the aneurysm models, each element represented a toroidal section of thrombus. We restricted our models to thrombi that exist within the aneurysmal cavity. We did not consider thrombi that encroached into the normal vessel lumen, thus all thrombi elements were within the 6.0 cm section of aneurysmal vessel. All thrombi were considered to be fixed to the underlying aneurysm wall.

Our models were computationally subjected to intraluminal pressures of 1.6×10^5 dynes/cm² (approximately 120 mm Hg). We computed displacements and Von Mises stresses at the midpoint and corners of each element. We used the MSC/pal 2 computer program,¹² running on Macintosh System 7.1, to perform our numerical calculations. The axisymmetric element formulation, stiffness matrix, matrix inversion routines, and stress and displacement reconstruction routines used in this program follow standard algorithms.^{1,13} The mathematical formulation along with other specific finite element information is documented elsewhere and will not be discussed here.^{12,13}

Investigations. We divided our study into five principle sections. We designed each section to systematically evaluate a specific geometric or material characteristic and determine how changes to this characteristic influence aneurysm wall stresses.

In our first set of analyses, we examined the importance of thrombus size. We initially covered

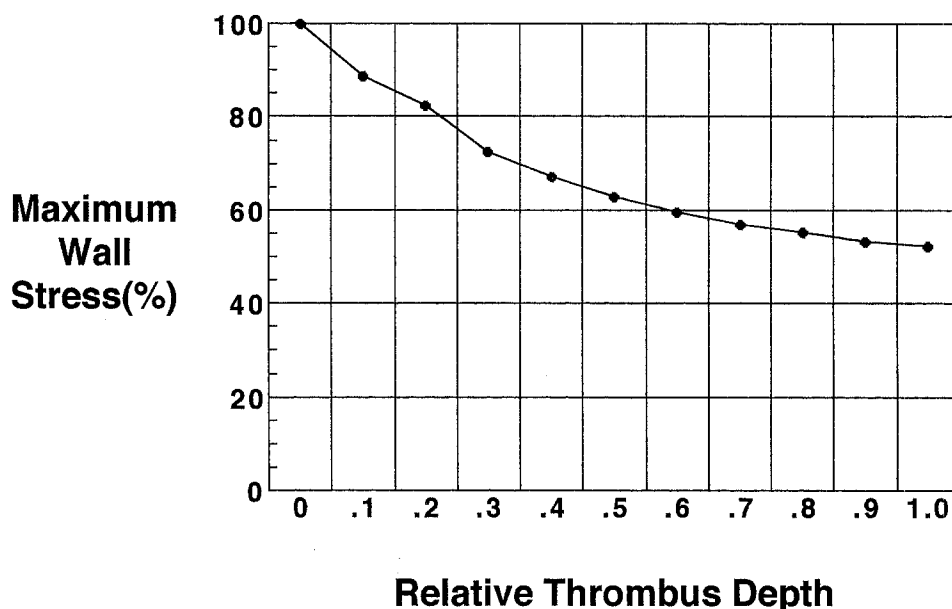


Fig. 2. Maximum wall stress decreases as the size of the intraluminal thrombus increases (maximum stress values are expressed as a percentage of the stresses found in an aneurysm with no intraluminal thrombus).

the inner 10% of our sinusoidal aneurysm model with intraluminal thrombus. In nine subsequent analyses, we increased the intraluminal thrombus thickness by 10% until thrombus filled the entire aneurysm cavity and reduced the flow channel to the dimensions of the inflow and outflow arteries. We did not allow thrombus to extend beyond the aneurysmal cavity, and we maintained patency in the normal vessel lumen. We calculated wall stresses and displacements for each incremental increase in thrombus size. We used constant shape, size, and material properties in the underlying aneurysm wall, and we used a constant elastic modulus of 0.2 MPa for the intraluminal thrombus.⁴

To evaluate the influence of thrombus strength, we varied the thrombus elastic modulus in our second set of analyses. We kept the size, shape, and material properties of the aneurysm wall constant and filled the entire aneurysm cavity with thrombus. We then calculated wall stresses and displacements as the thrombus elastic modulus increased from 0.01 MPa to 1.0 MPa.

We evaluated how aneurysm strength influences the effects of intraluminal thrombus in our third set of analyses. Using a constant 4.0 cm diameter sinusoidal aneurysm model, we calculated wall stresses and displacements as the aneurysm wall modulus increased from 1.0 MPa to 12.0 MPa. We assumed

that the aneurysmal cavity was filled with thrombus with a constant elastic modulus of 0.2 MPa. We also recalculated stresses for all models after reducing the thrombus modulus to 0.02 MPa.

In our fourth set of analyses, we evaluated how the effects of intraluminal thrombus vary as the size of the underlying aneurysm changes. We maintained constant elastic moduli for both the aneurysm wall (1.0 MPa) and the cavity-filling intraluminal thrombus (0.2 MPa). Starting with our initial 4.0 cm aneurysm model, we sequentially decreased the aneurysm diameter by 2.0 mm and constructed nine additional smaller aneurysm models. We preserved the sinusoidal contour in each of these aneurysms. We then calculated wall stresses and displacements for each model.

In our final analyses we examined the importance of inhomogeneity. We used two separate models for these analyses. In the first model we varied the material properties of the inner layer of the thrombus, allowing the elastic modulus to range from 0.01 MPa up to 2.0 MPa. We assigned an elastic modulus of 0.2 MPa to the remainder of the thrombus. In our second model we varied the material properties in the center of the thrombus, again allowing the elastic modulus to range from 0.01 MPa up to 2.0 MPa, while maintaining a constant modulus of 0.2 MPa for the remainder of the thrombus.

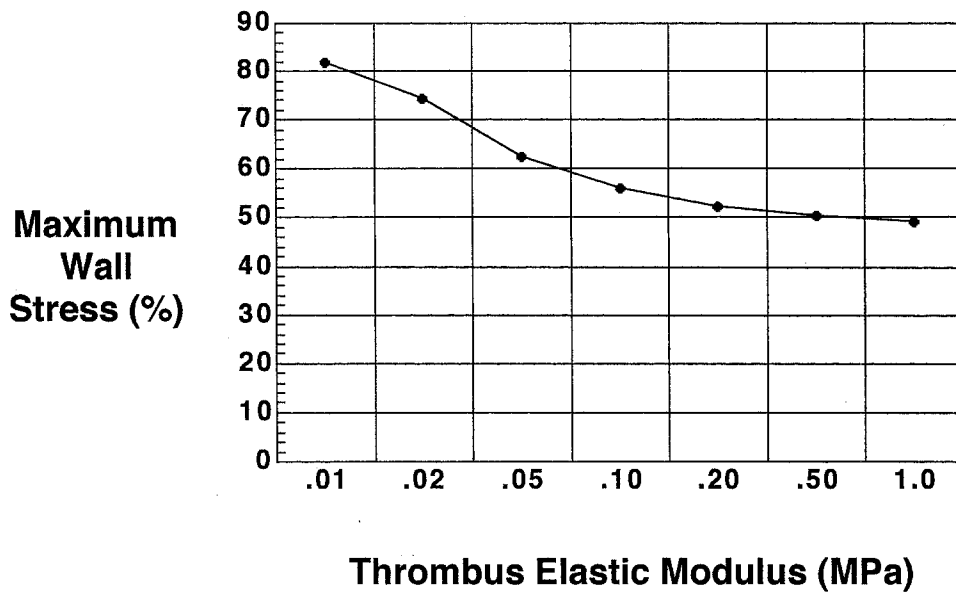


Fig. 3. Maximum wall stress decreases as the strength of the intraluminal thrombus increases (maximum stress values are expressed as a percentage of the stresses found in an aneurysm with no intraluminal thrombus).

To determine the effect of each geometric or material characteristic, we compared peak Von Mises stresses between the modified and unmodified aneurysms. Von Mises stresses are commonly used failure criteria that relate the deformational energy required to produce failure under uniaxial loading (the condition in which material properties are usually measured), with equivalent deformational energy under multiaxial loading. Von Mises stresses thus reflect local deformational energy, which in turn reflects the potential for material failure.¹²

RESULTS

Intraluminal thrombi reduced peak AAA wall stresses in all models. The degree of stress reduction was related to thrombus size as shown in Fig. 2. A maximum stress reduction of 48% occurred when thrombus with elastic modulus of 0.2 MPa filled the entire lumen of our 4.0 cm sinusoidal AAA. As the thrombus size decreased, so too did the degree of stress reduction. Thus thrombus filling the inner 10% of the aneurysm produced only an 11% reduction in peak wall stresses. Alternatively, changing the size of a small thrombus produced a greater change in peak stress than changing the size of a large thrombus. Increasing thrombus depth from 10% to 20% produced an additional 6.2% reduction in peak wall stress; increasing thrombus depth from 90% to 100% resulted in a decrease of only 1.2%.

The amount of stress reduction was also dependent on the material properties of the thrombus. Intraluminal thrombus with an elastic modulus of 0.01 MPa reduced peak wall stresses by 18%. An identical thrombus with an elastic modulus of 1.0 MPa generated a 51% reduction in peak wall stresses. Fig. 3 illustrates the relationship between stress reduction and thrombus material strength.

The elasticity of the underlying aneurysm wall also modulates the effects of intraluminal thrombus. These effects are greatest in pliant aneurysms; stiff aneurysm walls are relatively resistant to stress reduction from intraluminal thrombus. Stresses in our 4.0 cm diameter aneurysm model (containing an intraluminal thrombus with elastic modulus of 0.2 MPa) were reduced by 48% when the wall stiffness equaled 1.0 MPa. Stress was reduced by only 24% when wall stiffness was increased to 12.0 MPa. Decreasing the intraluminal thrombus elastic modulus by a factor of 10 (resulting in an elastic modulus of 0.02 MPa) resulted in stress reduction of only 6.8% when wall stiffness exceeded 12.0 MPa, and 25.6% for walls with a stiffness of 1.0 MPa. Fig. 4 illustrates the relationship between stress reduction and elastic properties of the underlying aneurysm wall.

Aneurysm size can significantly influence the amount of stress reduction. Peak wall stresses were reduced from 2.17×10^6 dyne/cm² to 1.13×10^6 dyne/cm² (48% reduction) when thrombus filled the

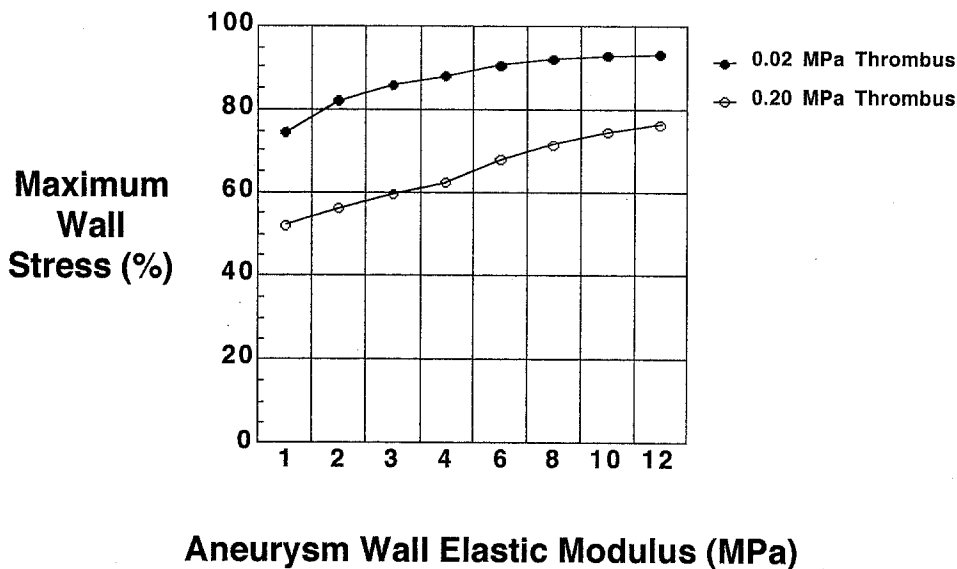


Fig. 4. Maximum wall stress increases as the aneurysm becomes stiffer. The intraluminal thrombus becomes less efficient at absorbing wall stresses. The magnitude of this effect depends on the elasticity of the intraluminal thrombus, as illustrated in the two separate curves (maximum stress values are expressed as a percentage of the stresses found in an aneurysm with no intraluminal thrombus).

aneurysmal cavity of a 4.0 cm aneurysm. Stresses decreased from 1.64×10^6 dyne/cm² to 1.13×10^6 dyne/cm² (31% reduction) in a similar 3.4 cm diameter aneurysm, and stresses decreased from 1.25×10^6 dyne/cm² to 1.12×10^6 dyne/cm² (11% reduction) in a 2.6 cm diameter aneurysm. Thus intraluminal thrombus not only produced a greater magnitude of stress reduction in larger aneurysms, but the relative decrease (percent reduction) in stress was greater. However, despite stress reduction from intraluminal thrombus, larger aneurysms uniformly exhibited greater wall stresses than smaller aneurysms. Thus although intraluminal thrombus was effective in reducing wall stress, its presence could not negate the effects of increasing aneurysm size.

Inhomogeneity produced only minimal changes in aneurysm wall stresses. When the elastic modulus for the inner layer of the thrombus was reduced to 0.01 MPa, peak stress grew from 1.13×10^6 dyne/cm² to 1.17×10^6 dyne/cm², a 3.5% increase. Stiffening the inner thrombus by increasing the modulus to 2.0 MPa resulted in peak wall stress of 1.07×10^6 dyne/cm², a 5.3% reduction. Inhomogeneity in the center of the thrombus also generated minimal changes in peak stress. Reducing the inner modulus to 0.01 MPa produced a 0.9% decrease in peak wall stress (1.13×10^6 dyne/cm² to 1.12×10^6 dyne/cm²), whereas increasing the modulus to 2.0 MPa

resulted in peak stresses of 1.15×10^6 dyne/cm², a 1.8% increase.

DISCUSSION

In-depth knowledge of the factors that influence aneurysm wall stress is critical for understanding aneurysm growth and rupture. Aneurysm failure occurs when local wall stresses exceed the material strength of wall tissues. This article furthers that knowledge by presenting our systematic examination of how intraluminal thrombi influence aneurysm wall stresses. It is apparent from our results that intraluminal thrombus can significantly reduce aneurysm wall stress. By reducing wall stresses, the thrombus also protects against aneurysm rupture. Thus intraluminal thrombus formation may be viewed as a natural protective response to aneurysmal dilatation.

The magnitude of the protective effect is dependent on several factors, including thrombus size, aneurysm size, thrombus properties, aneurysm stiffness, and inhomogeneities within the thrombus.

For a given size aneurysm, a large thrombus provides more protection than a small thrombus. This is particularly true if the thrombi are small relative to the volume of the aneurysmal cavity. As the size of the thrombi approach the size of the cavity, the increased protection from larger thrombi becomes smaller and smaller.

The protective effect of intraluminal thrombus is influenced by the size of the aneurysm. Larger aneurysm cavities can contain more thrombus, which in turn can "absorb" more stress. Thus the relative stress reduction is greater in larger aneurysms than it is in smaller aneurysms. Unfortunately, larger aneurysms have inherently greater wall stresses, and the protective effect of intraluminal thrombus is insufficient to reduce wall stresses to levels found in smaller aneurysms. Thus despite protection from intraluminal thrombus (if present), larger aneurysms have overall higher wall stresses and are at greater risk of rupture than are smaller aneurysms.

The protective effect of an intraluminal thrombus is also dependent on its elastic modulus. Stiffer thrombi stretch less and "absorb" more wall stress than pliant thrombi and are thus more efficient at reducing wall stresses. In addition, stiffer thrombi are often organized; their structural components not only make them stiffer, but structurally stronger. These stronger thrombi are less prone to failure and provide persistent protection to the aneurysm wall.

Biophysical studies indicate that the elastic modulus and the ultimate strength of thrombi vary widely.^{4,14} Both quantities depend on the conditions that are present at the time of thrombus formation, as well as events (including maturation) that follow formation. In general, well-organized mature thrombi have greater ultimate strength and better elastic properties than new or poorly organized thrombi.¹⁴ Unfortunately, it is difficult to determine the elastic modulus or failure limit of an intraluminal thrombus. This in turn makes it difficult to clinically assess the impact of a specific thrombus. Care must be taken not to overestimate the material characteristics of thrombi in individual aneurysms.

Our study clearly demonstrates that the material properties of the aneurysm influence the degree of stress reduction produced by intraluminal thrombus. The greatest stress reductions occur when elastic properties of thrombus approach those of the aneurysm wall. This is because a pliant thrombus must deform before it can absorb a significant amount of stress. If a pliant thrombus is contained within a stiff aneurysm, there will be insufficient room for deformation and the thrombus will be unable to absorb a significant amount of stress. The "unabsorbed" forces will then be transmitted to the aneurysm wall, where they will act to increase wall stress. If the thrombus is stiffer (or the wall more pliant), deformation can occur and the thrombus can absorb stress and protect the underlying wall.

Inhomogeneity within a thrombus can alter the

magnitude of stress reduction. However, the effect of inhomogeneity is relatively small. Changing the elastic modulus of our inhomogeneity from 0.01 MPa to 2.0 MPa (two orders of magnitude) produced only small changes in peak wall stresses (less than 10%). Overall thrombus size and average material properties are much more important in determining stress reductions. This implies that it is not necessary to obtain detailed knowledge of thrombus material properties for estimating stress reductions. However, it is important to note that although inhomogeneities have a minimal effect on wall stresses, they can generate significant stress distortions within the thrombus itself. Such stress distortions can produce failure within the thrombus and, in destroying the thrombus, would have an indirect effect on aneurysm wall stresses.

Our computational results complement clinical findings. Pillari et al.¹⁵ observed that rupture was unusual in aneurysms that had intraluminal thrombi, but aneurysmal dilatation and rupture rates increased as the amount of thrombus decreased. Pillari et al. also found that 90% of intraluminal thrombi cover the ventral aorta (either as a ventral crescent or concentric thrombus) and suggested that protective thrombi may explain why ventral intraperitoneal aneurysm ruptures are rare. Our results demonstrate that intraluminal thrombus can provide significant protection to aneurysmal aortic walls. By reducing wall stresses, intraluminal thrombi protect against aneurysm growth and rupture.

Our findings are best summarized in the following: (1) All other things being equal, an aneurysm with a large intraluminal thrombus will have lower wall stresses than a similar aneurysm with little or no thrombus. (2) More organized and stiffer thrombi provide greater reductions in wall stress than poorly organized and weak thrombi. (3) Larger aneurysms experience greater reductions in wall stress than smaller aneurysms. However, the inherently greater stresses present in the walls of large aneurysms negate the protective effect of intraluminal thrombus. Large aneurysms with large intraluminal thrombi have greater wall stresses than small aneurysms with small intraluminal thrombi. (4) Intraluminal thrombi are of greatest benefit in pliant aneurysms. Stiffer aneurysms experience less stress reduction. (5) Material inhomogeneities produce insignificant changes in the stress reduction produced by intraluminal thrombus.

Limitations. Our purpose in conducting this study was to obtain qualitative information on how intraluminal thrombi influence aneurysm wall

stresses. We did not intend to analyze the detailed quantitative behavior of specific aneurysms. To this end, we used a simplified and idealized aneurysm model. In reality, aneurysms are not axisymmetric structures that have smooth, homogeneous, and isotropic walls, and they do not exhibit linear elastic deformation. These simplifying assumptions make our results quantitatively limited, and it would be unwise to extract detailed quantitative information from our results. Thus we would be remiss in stating that an intraluminal thrombus will reduce wall stress in 4.0 cm aneurysms by 52%. The actual degree of stress reduction may be somewhat more or somewhat less. However, we can confidently assert that intraluminal thrombus will significantly reduce wall stress, and that the amount of stress reduction will depend on the size of the intraluminal thrombus.

The static axisymmetric models used in our analyses do not allow us to analyze pulsatile flow effects, nor do they allow us to evaluate asymmetric structures such as eccentric thrombi or asymmetric aneurysms. Furthermore, natural aneurysms exhibit wide variation in shape, size, material properties, and loading conditions (including vascular tethering). Many of the detailed properties of individual aneurysms are difficult, if not impossible, to measure in living subjects. This makes it imprudent to use our results to predict the stresses that are actually present in individual aneurysms. In particular, it would be unwise to use our qualitative results in making quantitative decisions (such as risk of aneurysm rupture) for individual patients. Future studies, using more generalized three-dimensional models, may address more complicated structures and allow analysis of saccular and asymmetric aneurysms.

Finally, this study focuses on how intraluminal thrombus affects aneurysm wall stress. Our findings indicate that thrombus may be protective and reduce the risk of aneurysm rupture. Such benefit must be weighed against the risks of intraluminal thrombus, such as embolization.

CONCLUSION

Intraluminal thrombus can effectively reduce wall stress in abdominal aortic aneurysms. The effect is

greatest for large, well-organized thrombi contained within pliant aneurysms, but even small thrombi can effectively reduce wall stress.

REFERENCES

1. Zienkiewicz OC. The finite element method. 3rd ed. London: McGraw-Hill, 1977.
2. Stringfellow MM, Lawrence PF, Stringfellow RG. The influence of aorto-aneurysm geometry upon stress in the aneurysm wall. *J Surg Res* 1987;42:425-33.
3. Mower WR, Baraff LJ, Sneyd J. Stress distributions in vascular aneurysms: factors affecting risk of aneurysm rupture. *J Surg Res* 1993;55:155-61.
4. Inzoli F, Boschetti F, Zappa M, Longo T, Fumero R. Biomechanical factors in abdominal aortic aneurysm rupture. *Eur J Vasc Endovasc Surg* 1993;7:667-74.
5. Horejs D, Gilbert PM, Burstein S, Vogelzang RL. Normal aortoiliac diameters by CT. *J Comput Assist Tomogr* 1988; 12:602-3.
6. Dixon AK, Lawrence JP, Mitchell JRA. Age-related changes in the abdominal aorta shown by computed tomography. *Clin Radiol* 1984;35:33-7.
7. Rizzo RJ, McCarthy WJ, Dixit SN, Lilly MP, Shevely VP, Flinn WR, Yao JST. Collagen types and matrix protein content in human abdominal aortic aneurysms. *J Vasc Surg* 1989; 10:365-73.
8. Bergel DH. The static elastic properties of the arterial wall. *J Physiol* 1961;156:445-57.
9. Learoyd BM, Taylor MG. Alterations with age in the viscoelastic properties of human arterial walls. *Circ Res* 1966;18: 278-92.
10. Carew TE, Vaishnav RN, Patel DJ. Compressibility of the arterial wall. *Circ Res* 1968;23:61-8.
11. Hayashi K. Experimental approaches on measuring the mechanical properties and constitutive laws of arterial walls. *J Biomech Eng* 1993;115:481-8.
12. MSC/pal 2: Advanced Stress and Vibration Analysis, Reference Manual: Apple Macintosh Version. Los Angeles: MacNeal-Schwendler Corporation, 1989.
13. Huebner KH. The finite element method for engineers. New York: John Wiley and Sons, 1975.
14. Hartert H, Schaefer JA. The physical and biological constants of thrombelastography. *Biorheology* 1962;1:31-9.
15. Pillari G, Chang JB, Zito J, Cohen JR, Gersten K, Rizzo A, Bach AM. Computed tomography of abdominal aortic aneurysm. *Arch Surg* 1988;123:727-32.

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