# Mechanical wall stress in abdominal aortic aneurysm: Influence of diameter and asymmetry

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*Purpose:* Risk for rupture of an abdominal aortic aneurysm is widely believed to be related to its maximum diameter. From a biomechanical standpoint, however, risk is probably more precisely related to mechanical wall stress. Many abdominal aortic aneurysms are asymmetric (for example because of anterior bulging with posterior expansion limited by the vertebral column). The purpose of this work was to investigate the effect of maximum diameter and asymmetric bulge on wall stress.

*Methods:* Three-dimensional computer models of abdominal aortic aneurysms were generated. In one protocol, maximum diameter was held constant while bulge shape factor was varied. The shape factor took into account the asymmetric shape of the bulge. In a second protocol, the shape of the aneurysmal wall was held constant while maximum diameter was varied. Wall stress was computed in each instance with a commercial software package and assumption of physiologic intraluminal pressure.

*Results:* Both maximum diameter and the shape factor were found to have substantial influence on the distribution of wall stress within the aneurysm. In some instances the maximum stress occurred at the midsection, and in others it occurred elsewhere. The magnitude of peak stress acting on the aneurysm increased nonlinearly with increasing maximum diameter or increasing asymmetry.

*Conclusions:* Our computer models showed that the stress within the wall of an abdominal aortic aneurysm and possibly the potential for rupture are as dependent on aneurysm shape as they are on maximum diameter. This information may be important in determining severity of individual abdominal aortic aneurysms and in improving understanding of the natural history of the disease. (J Vasc Surg 1998;27:632-9.)

Abdominal aortic aneurysm (AAA) disease occurs among approximately 2% of the elderly population, and the incidence apparently is increasing.<sup>1</sup> Rupture of an AAA is currently ranked as the 13th most common cause of death in the United States.<sup>2</sup> Surgical repair of AAA is performed to prevent death from rupture and is associated with acceptable but not insignificant morbidity and mortality rates.<sup>3-5</sup> It is important to determine when during the natural history of an aneurysm, risk for rupture justifies the operation and its potential attendant complications

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and expense.<sup>6,7</sup> At present the decision for elective repair of an AAA is usually based on the maximum diameter of the aneurysm. Several other anatomic variables have been proposed for use as predictors of aneurysm severity.<sup>1,6,8</sup> These criteria are quite crude and do not take into account certain important characteristics of individual aneurysms. For example, AAAs with the same maximum diameter may have differences in shape, wall thickness, or mechanical properties that affect their propensity for growth and rupture. Thus an operation based on a 5 cm critical diameter or other similar criterion may be unjustified (low risk for rupture) or too late (rupture at less than 5 cm) for a particular patient.<sup>9-11</sup>

From a biomechanical perspective, the proper definition of the critical state of an AAA is that at which the mechanical stress within the aneurysmal wall exceeds the tensile strength of the tissue. We have reported on the tensile strength of aneurysmal tissue,<sup>12,13</sup> and there have been efforts to estimate wall stress within AAAs.<sup>14-17</sup> The purpose of this

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**Fig. 1.** Representative, three-dimensional, virtual AAA model (*top*) and midsectional cross section (*bottom*).  $r_p$  and  $r_a$ , maximum posterior and anterior wall dimensions, respectively, measured from the *dashed longitudinal line* defined by the centers of the two undilated ends.  $r_p + r_a =$  maximum diameter. Inflection points (*IP*) are defined as points on the AAA surface at which the local AAA wall shape changes from concave outward to concave inward and are indicated here only for the anterior and posterior surfaces. The angular circumferential coordinate  $\theta$  is defined as shown.

study was to investigate the separate effects of AAA bulge shape and diameter on wall stress distribution in AAAs. Most AAAs are not axisymmetric.<sup>18,19</sup> For example, limitation of posterior expansion caused by the vertebral column might result in preferential anterior expansion of the aneurysmal wall and an asymmetric configuration. An asymmetric shape might greatly influence stresses on the wall of an AAA and might be as important a clinical consideration as aneurysmal diameter.

We performed computational analyses using three-dimensional computer models (or virtual AAAs) wherein maximum diameter and degree of asymmetry were individually controlled. Our com-





**Fig. 2.** Three-dimensional models of AAA represents the two extreme cases studied for each protocol. Protocol 1, variation of asymmetry parameter  $\beta$  from 1.0 (axisymmetric) to 0.3 while maximum diameter ( $D_{max}$ ) was held constant at 6 cm. Protocol 2, variation of maximum diameter from 4 cm to 8 cm while  $\beta$  was held constant at 0.4. Three intermediate cases were studied between the two extremes for each protocol.

puter simulations showed that asymmetric shape is an important factor affecting mechanical wall stress within an AAA and that consideration of maximum diameter alone may be insufficient as an estimation of the severity of an individual AAA.

# METHODS

Geometric modeling. Computer models of hypothetical AAAs were generated with commercial software (Pro-Engineer v. 16.0; Parametric Technology Waltham, Mass.) such that overall length was 12 cm and cross section at any axial position was circular, as has been shown to be typical.<sup>8,20</sup> A representative model is shown in Fig. 1. Because wall thickness was not studied in this investigation, it was assumed to be uniform throughout at 1.5 mm. The posterior and anterior wall profiles of the AAA models were generated from normal Gaussian distribution (bell curves). Important locations on the wall of the virtual AAA, particularly the inflection points of the surface, are shown in Fig. 1. These are locations anywhere on the wall where the surface of the aneurysm changes from concave outward to concave inward.

For a separate investigation of the effect of maximum diameter and asymmetry on wall stress distribution in an AAA, the virtual AAAs were generated according to two protocols (Fig. 2). In one protocol maximum diameter was kept constant at 6 cm while an asymmetry parameter  $\beta$  was varied, where  $\beta = r_p/r_a$ . As shown in Fig. 1,  $r_p$  is maximum posterior wall dimension, and  $r_a$  is maximum anterior wall dimension. It can be seen that  $r_p + r_a = maximum$  diameter. The anatomy of an AAA is usually such that  $r_p < r_a$  ( $\beta < 1$ ) because of the proximity of the posterior wall to the vertebral column. Thus computer models were generated with  $\beta$  that varied from 0.3 to 1.0. A value of 1.0 for  $\beta$  represents an axisymmetric AAA with equal anterior and posterior bulging; a value of 0.3 represents a highly asymmetric AAA with a highly preferential anterior bulge. In the other protocol, models were generated for a constant asymmetry ( $\beta = 0.4$ ), and maximum diameter varied from 4 cm to 8 cm. The diameter of the undilated aorta ranged from 2.1 cm to 2.4 cm in the models.

Ten total virtual AAAs were constructed, five for each protocol. The two extreme cases for each protocol are shown in Fig. 2. All AAA models have at least two planes of symmetry, namely, the transverse plane and the median plane. Because of the transverse plane of symmetry, there are two inflection points in the AAA profile shape, one above the midsection and one below (Fig. 1).

**Finite element model.** The complex shape of the aneurysmal wall precludes a solution for the wall stress by means of straightforward analytic techniques. Instead we used a technique<sup>14-16,21</sup> known as finite element analysis for computation of the wall stresses in each virtual AAA. In this technique, a body of complex shape is divided into smaller, simpler shaped elements. The stresses over the individual elements are computed, and the solution is patched together to yield the stress distribution for the entire complex body. Specific technical details regarding finite element analysis may be found elsewhere.<sup>21</sup>

The three-dimensional AAA models were imported into the finite element software package ANSYS (version 5.3; Ansys, Houston, Pa.) and discretized into small elements with a linearly elastic, quadrilateral shell element. The number of elements ranged from 3200 to 3800 depending on the shape and size of the virtual AAA. The elastic modulus (500 Newtons/cm<sup>2</sup>) used in the analysis was obtained by means of previous tensile tests of AAA tissue performed by us.<sup>12,13</sup> It has been shown that vascular tissue is nearly incompressible,<sup>22</sup> and we used a Poisson ratio of 0.49.

Static analysis was performed. A peak systolic arterial pressure load (120 mm Hg [1.6 Newtons/cm<sup>2</sup>]) was applied uniformly on the internal surface of the AAA models. Shear stresses caused by flowing blood were not considered because they are shown to be small in magnitude compared with stresses caused by distention of the wall.<sup>16,23</sup> The outer surface of the AAA model was taken as load free, and the two ends were held fixed in the axial direction. This longitudinal constraint took into account tethering of the aorta by the surrounding connective tissue and the vertebral and other collateral arteries. To represent the complex stress distribution in the wall of each virtual AAA, von Mises stress distribution<sup>16</sup> was computed and inspected for each simulation. The von Mises stress is derived from the distortion energy used in studies of material failure. It is a function of the three principle stresses in the body of an AAA.<sup>24</sup>

## RESULTS

The three-dimensional distribution of mechanical wall stress for each of the ten virtual AAA models is shown in two views in Fig. 3. The results for protocol 1 (varying asymmetry) are shown in Fig. 3, *A*, and those for protocol 2 (varying maximum diameter) are shown in Fig. 3, *B*. The effects of the asymmetry parameter  $\beta$  and maximum diameter on the circumferential variation of wall stress around the AAA midsection (along the dashed curve in Fig. 1) is shown in Fig. 4. The maximum stress at the AAA midsection occurred on the posterior surface in all models studied.

The effects of asymmetry and maximum diameter on the longitudinal variation of wall stress along the anterior surface of the virtual AAA are shown in Fig. 5. Both parameters were found to have similar effects. Increasing the diameter or asymmetry of an AAA causes an increase in wall stress at the inflection points of the profile shape (Fig. 1) while causing a decrease in stress at the midsection. The effects of asymmetry and maximum diameter on longitudinal variation of wall stress along the posterior surface of a virtual AAA are shown in Fig. 6. The effects are similar to those found for the anterior surface but with the following differences. As the aneurysm becomes more asymmetric, the greatest stress on the posterior surface relocates from the inflection points to the midsection (Fig. 6, A). Conversely, as the aneurysm enlarges, the maximum stress relocates from the midsection to the inflection points (Fig. 6, B).

When an AAA is small, the maximum wall stress occurs on the posterior wall at the midsection (compare Figs. 5, B and 6, B). As the AAA enlarges, the stress on the posterior wall at the midsection remains elevated, but the maximum stress occurs at the inflection points on the anterior surface. The peak stress within the virtual AAA was found to increase nonlinearly with increasing diameter as well as with increasing asymmetry (Fig. 7).



**Fig. 3.** Distribution of mechanical wall stress in all ten virtual AAAs. **A**, Models for protocol 1 (varying asymmetry). **B**, Models for protocol 2 (varying maximum diameter). Each model is shown in two views. The *left columns* of both A and B provide a view of the anterior, left lateral surface of the virtual AAA. The *right column* provides a view of the posterior, right lateral surface. The sketch at the top of each column shows the vertebral column and provides anatomic reference. The magnitude of the mechanical wall stress for both protocols is given on the individual color scales in Newtons/cm<sup>2</sup> (note: 1 Newton/cm<sup>2</sup> = 10<sup>5</sup> dynes/cm<sup>2</sup>).

#### **Circumferential Distribution of Wall Stress at Midsection**



Fig. 4. Effect of asymmetry parameter  $\beta$  (A) and maximum diameter (B) on circumferential distribution of wall stresses at the midsection of the AAA (along dashed curve in Fig. 1). The  $\theta$  coordinate is as defined in Fig. 1. A position of  $\theta = 0$  degrees corresponds to the posterior surface, and ±180 degrees corresponds to the anterior surface. For this and subsequent Figures, a decrease in  $\beta$  corresponds to an increase in asymmetry.

## DISCUSSION

Risk for rupture of AAA is widely believed to be associated with maximum diameter. It has been observed clinically, however, and autopsy studies show that some large aneurysms do not rupture while some small aneurysms do.9 There is a need for a better definition of the severity of AAA. Aneurysm rupture is caused by a gross mechanical failure of the aortic wall and occurs when the acting mechanical wall stress exceeds the strength of the tissue. We believe that knowledge of the wall stresses in AAA would provide clinicians with a more accurate estimate of the likelihood of rupture of an individual aneurysm. Our study showed that the stress within the wall of an AAA and possibly its propensity for rupture depend on the shape and the diameter of the aneurysm.

To our knowledge, this was the first study to investigate the effects of asymmetry on three-dimensional stress distribution in the wall of AAAs. Previous

#### Longitudinal Distribution of Wall Stress on Anterior Surface



0 2 4 6 8 10 12 Axial position, cm proximal neck Fig. 5. Effect of asymmetry parameter  $\beta$  (A) and maximum diameter (B) on longitudinal distribution of wall stresses along the anterior surface of an AAA. Axial positions of z = 0 cm and z = 12 cm correspond to the supe-

rior and inferior undilated ends of the AAA; z = 6 cm cor-

0

responds to the midsection.

consideration of AAA wall stresses have used the law of Laplace,<sup>19</sup> or investigators assumed axisymmetric geometry.<sup>14-17</sup> The law of Laplace is often incorrectly used because in its most common forms, it is applicable only to cylinders or spheres. It is thus insufficient to provide estimates of the complex stress distributions in AAA. Using the law of Laplace, one would predict greatest wall stresses at the midsection of each of the virtual AAAs studied because diameter was maximum there. Our computer models showed that this is usually not the case (Figs. 3, 5, and 6). We showed here that stress distribution in asymmetric AAA is markedly different from that in axisymmetric AAA. Previous models with axisymmetric AAA shapes (for which  $\beta = 1$ ) would fail to describe important variations of stresses along the aneurysmal wall caused by asymmetry (from preferential bulging, for example). Nonetheless, if their limitations are kept in mind, previous models have provided important insight into the biomechanical features of AAA.

#### Longitudinal Distribution of Wall Stress on Posterior Surface

#### A. Effect of asymmetry



Fig. 6. Effect of asymmetry parameter  $\beta$  (A) and maximum diameter (B) on longitudinal distribution of wall stresses along the posterior surface of an AAA. The longitudinal axis is the same as in Fig. 5.

Stringfellow et al.<sup>14</sup> used finite element analysis to investigate the differences in stress distribution for spherical versus cylindrical AAAs. Although they performed their analysis on axisymmetric shapes, they appear to have been the first to consider shape effects on AAA stress distribution. Their results showed that circumferential stress in the walls of cylindrical aneurysms is greater than that in spherical aneurysms and that longitudinal stress is unaffected. One possible limitation of the investigation by Stringfellow et al., however, was use of a sharp junction between the AAA and undilated aorta. Our geometric modeling techniques allowed a smoother transition, avoiding unrealistic stress concentrations. Inzoli et al.<sup>16</sup> also used an axisymmetric AAA model to demonstrate the effects of maximum diameter and presence of intraluminal thrombus and atherosclerotic plaque within the wall. In agreement with our results (Fig. 7), they found that peak wall stress increased with increasing maximum diameter. They also showed that the presence of a calcified atherosclerotic plaque caused elevated stress concentrations and an increase in peak stress, whereas intralu-



**Fig. 7.** Effect of asymmetry parameter  $\beta$  (*bottom axis*) and maximum diameter (*top axis*) on magnitude of peak stress within a virtual AAA. Both increasing diameter and increasing asymmetry (decreasing  $\beta$ ) cause a nonlinear increase in peak stress.

minal thrombus caused a decrease in peak stress by up to 30%. We suggested the mechanically protective consequence of intraluminal thrombus in previous experimental studies.<sup>25</sup> Mower et al.<sup>15</sup> demonstrated with an axisymmetric model that AAA wall stress increases in proportion to maximum diameter and in inverse proportion to wall thickness. As we did in this study, Mower et al. determined that maximum stress in axisymmetric AAA models occurred at the inflection points of the profile shape. Elger et al.17 showed that the shape of an axisymmetric AAA influences stress distribution. They suggested that maximum wall stress is a function of the curvature of the wall profile. We extended these previous studies by considering aneurysms that were not axisymmetric but had more realistic asymmetric shapes produced by preferential or nonuniform bulging.

Our results indicated that for small AAAs ( $\leq 5$  cm) maximum stress occurs on the posterior wall but for larger AAAs peak stress occurs on the anterior surface. Mechanical failure of aortic tissue leads to AAA rupture and occurs when local wall stress exceeds local wall strength. We have reported on wall strength of AAA tissue<sup>12,13</sup> and have found that the strength of the tissue near the neck or undilated ends of an AAA are greater than that in the midsection, where diameter is maximum (unpublished results). Although the stresses are maximum on the anterior surface for larger AAAs, the actual propensity for rupture for that or any other surface depends

# Effect of Diameter and Asymmetry on Peak Wall Stress

on the comparative local value of wall strength. Because wall strength may be lower at the midsection and stresses are greatest on the posterior surface (Fig. 4), it is possible that rupture would occur on this surface before it would occur on the anterior surface, where stresses are actually higher. Autopsy studies have shown that there is no clear predilection for site of rupture of AAA, but it seems to occur most frequently along the posterior surface.<sup>9</sup> To our knowledge, no study has been performed to correlate diameter with rupture site. Comparison of our results with those of such a study would allow more rigorous interpretation of the importance of local wall stress in AAA rupture.

The relative importance of maximum diameter and  $\beta$  can be demonstrated with inspection of Figs. 4 through 7. For example, Fig. 4, A shows that the value of stress on the midsection of the posterior wall is increased by more than 85% when the AAA changes shape from axisymmetric ( $\beta = 1$ , stress 15 Newtons/cm<sup>2</sup>) to highly asymmetric ( $\beta = 0.3$ , stress 28 Newtons/cm<sup>2</sup>) with maximum diameter constant. Fig. 4, B shows that stress at the same site is increased by only 33% because of an increase in maximum diameter from 4 cm (stress 18 Newtons/cm<sup>2</sup>) to 8 cm (stress 24 Newtons/cm<sup>2</sup>) with  $\beta$  constant. Fig. 7 demonstrates that the value of peak wall stress is increased by 137% with an increase in maximum diameter from 4 cm (stress 19 Newtons/cm<sup>2</sup>) to 8 cm (stress 45 Newtons/cm<sup>2</sup>) but is increased by 45% with an increase in asymmetry from 1.0 (stress 22 Newtons/cm<sup>2</sup>) to 0.3 (stress 32 Newtons/cm<sup>2</sup>).

According to the critical diameter criterion often used to guide decisions for elective AAA repair, all AAAs of the same diameter have the same likelihood of rupture. Our results show that this is probably not the case. For example, Fig. 6, A depicts results for five different virtual AAAs with a diameter of 6 cm. The critical diameter criterion would suggest that each of these AAAs has an equal predilection for rupture. However, the stress on the midsection of the posterior wall of a highly asymmetric AAA ( $\beta =$ 0.3) is twice that of an axisymmetric AAA ( $\beta =$  1.0). If the strength of the aortic wall were the same for both, the asymmetric aneurysm would be more likely to rupture.

The finite element method provides a means to inspect the stress distribution in individual AAA, and this can lend itself as a powerful clinical and research tool. With a stress map, it was possible to study the individual effects of aneurysmal diameter and bulge shape on stress distribution in virtual AAAs. Similar techniques could be used as a reliable, noninvasive

method to evaluate the severity of actual individual AAAs. This estimate of severity would be based on biomechanical wall stresses calculated with threedimensional reconstructions from computed tomographic scans or magnetic resonance images<sup>26</sup> and could be an important clinical tool to guide surgeons in decisions about elective repair of AAA. Studying stress distribution in real AAAs would be a useful clinical research tool by providing means to study the biologic consequences of mechanical stresses. For example, as pointed out by Mower et al.,15 vasa vasorum and other small blood vessels in regions of increased stress may tend to be compressed and not provide nutrition to the vessel wall. Thus sites of AAA with increased wall stress may undergo weakening with impaired repair or remodeling mechanisms. Increased local stresses or stress gradients likewise may regulate gene expression by the aortic wall, which would have an influence on natural history.

**Limitations.** In this analysis it was assumed that the AAA wall is homogeneous, isotropic, and linearly elastic and that it undergoes small strains, which is not the case.<sup>12,27,28</sup> However, the analysis provided a first approximation of the effects of AAA asymmetry and diameter, which was the goal of this study. We also assumed that the mechanical properties and thickness of the AAA wall are uniform over the surface. This is likely not realistic because of localized calcifications, for example, and variations of these parameters should be accounted for when studying stresses in real AAA. Though the same undilated aortic diameter was not used for all models, the slight variation was not enough to cause significant differences in stress distribution.

We provide what we believe is the first demonstration that asymmetry of the AAA bulge is important in considering mechanical wall stresses. However, all the virtual AAAs studied had at least two planes of symmetry, the median plane and the transverse plane. Although we used more realistic shapes than in previous studies, actual AAAs generally have even more complex shapes<sup>18,19</sup> and have no planes of symmetry. Future analyses should evaluate the effect of other asymmetries on mechanical wall stresses in AAA.

**Conclusion.** The asymmetry of an AAA, in addition to aneurysm diameter, is an important determinant of mechanical wall stress. Despite the critical diameter criterion often used to assess severity of an AAA, aneurysms with the same diameter may not necessarily have the same propensity for rupture. This information may be important in understanding the natural history of AAA and in the clinical management of this disease.

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