Wall stress distribution on three-dimensionally reconstructed models of human abdominal aortic aneurysm

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Purpose: Abdominal aortic aneurysm (AAA) rupture is believed to occur when the mechanical stress acting on the wall exceeds the strength of the wall tissue. Therefore, knowledge of the stress distribution in an intact AAA wall could be useful in assessing its risk of rupture. We developed a methodology to noninvasively estimate the in vivo wall stress distribution for actual AAAs on a patient-to-patient basis.
Methods: Six patients with AAAs and one control patient with a nonaneurysmal aorta were the study subjects. Data from spiral computed tomography scans were used as a means of three-dimensionally reconstructing the in situ geometry of the intact AAAs and the control aorta. We used a nonlinear biomechanical model developed specifically for AAA wall tissue. By means of the finite element method, the stress distribution on the aortic wall of all subjects under systolic blood pressure was determined and studied.
Results: In all the AAA cases, the wall stress was complexly distributed, with distinct regions of high and low stress. Peak wall stress among AAA patients varied from 29 N/cm^2 to 45 N/cm^2 and was found on the posterior surface in all cases studied. The wall stress on the nonaneurysmal aorta in the control subject was relatively low and uniformly distributed, with a peak wall stress of 12 N/cm^2. AAA volume, rather than AAA diameter, was shown by means of statistical analysis to be a better indicator of high wall stresses and possibly rupture.
Conclusion: The approach taken to estimate AAA wall stress distribution is completely noninvasive and does not require any additional involvement or expense by the AAA patient. We believe that this methodology may allow for the evaluation of an individual AAA's rupture risk on a more biophysically sound basis than the widely used 5-cm AAA diameter criterion. (J Vasc Surg 2000;31:760-9.)

The ability to evaluate reliably the susceptibility of a particular abdominal aortic aneurysm (AAA) to rupture could vastly improve the clinical treatment of patients with AAAs. Biomechanical approaches toward assessing the likelihood of AAA rupture have been previously proposed.[1-6] The basic premise of the biomechanical approach is that AAA formation and enlargement is accompanied by an increase in wall stress (internal forces per unit area), a decrease in the ability of the material of the wall to withstand these stresses (ie, a decrease in the wall's failure strength), or both. For example, a computational investigation by our group with hypothetical three-dimensional (3D) models demonstrated that the peak wall stress for a nonaneurysmal aorta is approximately 9 N/cm^2, whereas that for a 4-cm AAA and an 8-cm AAA is approximately 23 N/cm^2 and 45 N/cm^2, respectively (*N*, Newtons; 1 N/cm^2 = 10^5 dynes/cm^2 = 10 kilo Pascals [kPa] = 1.48 psi).[5] Ex vivo mechanical testing of healthy and aneurysmal abdominal aortic wall specimens in our laboratory showed that the failure strength of the aortic wall is reduced from 121 N/cm^2 in nonaneurysmal aorta to 65 N/cm^2 in aneurysmal aorta.[6,7] It is likely, therefore, that AAA rupture occurs at some point during aneurysmal growth, when the increasing wall stress exceeds the diminishing failure strength of the degenerating aortic wall. The moment at which wall stress exceeds its own strength likely varies from

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patient to patient and is not necessarily related to AAA diameter alone.[3-5,8,9] Thus, the ability to reliably predict acting wall stress and failure strength for a particular AAA could allow for a reliable evaluation of its propensity to rupture.

Although it is difficult to determine the failure strength of a particular AAA without destructively testing a piece of tissue excised from it, it is possible to estimate noninvasively the existing wall stress by using state-of-the-art 3D reconstruction and computational techniques. We believe that the knowledge of mechanical wall stress alone would lead to an improved assessment of a particular AAA's risk of rupture.

The goal of this study was to develop and demonstrate a methodology to determine the wall stress distribution in intact AAAs on a patient-specific basis. This technique uses the actual 3D geometry of the AAA under analysis and a realistic, nonlinearly elastic biomechanical model. With computational mechanics techniques, the distribution of stress on the aortic wall of six patients with aneurysms and one control patient without an aneurysm was determined. The possible correlation of wall stress with various clinically measurable factors was evaluated.

METHODS

Three primary pieces of information are required to perform stress analysis of an AAA: (1) the geometry of the AAA being evaluated; (2) the mathematical model that characterizes the biomechanical behavior of aneurysmal aortic tissue; and (3) the physiological forces and constraints acting on the AAA wall. The accuracy of the computed wall stress distribution depends on how rigorously these three components are defined in the analysis.

Study subjects. Seven patients at the University of Pittsburgh Medical Center were the subjects in this study. Six of the patients had an AAA and were awaiting repair. One control subject without an aneurysm, who was being evaluated for an unrelated condition, was included for comparison purposes.

Abdominal aortic aneurysm geometry. The geometry of an actual human AAA is too complex to be reliably approximated with hypothetical representations, as has been done in previous stress analyses.[1-5] For clinically meaningful and accurate results, it is necessary to use the actual "irregular" geometry of an individual AAA. In this study, spiral computed tomography (CT) data was used to create 3D reconstructions of the infrarenal aorta of study subjects. Because spiral CT is routinely performed on AAA patients scheduled for repair, collection of this information involved no extra participation by the study subjects. Abdominal CT scanning was performed with a General Electric spiral CT scanner (model Hi Speed Advantage). Before scanning, 150 mL of standard nonionic contrast was administered at 4 mL/s. Images were obtained during a single sustained breath hold by the patient to reduce respiratory-induced motion and associated artifact. The slice thickness (collimation) was between 3 to 5 mm, with a helical pitch of 1.5. After the raw spiral CT data were obtained, individual cross-sectional image slices were generated at 2- to 3-mm slice spacing along the infrarenal aorta.

Digital files containing the cross-sectional images from immediately distal to the renal arteries to immediately proximal to the iliac bifurcation were imported into the public domain image processing software, NIH-Image (version 1a, US National Institutes of Health). The boundary of the wall was identified with grayscale thresholding and semiautomated edge detection.[10] The thickness of the aortic wall was not identifiable in the CT images because of artifacts and image resolution. Therefore, only the outer wall of the infrarenal aorta was identified. The spatial coordinates of approximately 60 discrete points along the wall boundary were recorded on each cross section. From this, a point cloud representing discrete points on the AAA wall was obtained (Fig 1, *A*). From the point cloud, a mathematical representation of the AAA wall surface was generated with the public domain triangulation software NUAGES (version 4.1, INRIA, Sophia Antipolis Cedex, France). The resulting aortic wall surface contained sharp corners, which were artifacts from the imaging and image processing (Fig 1, *B*). Such sharp corners would result in artificial stress concentrations in stress analysis and lead to erroneous interpretations. A previously described surface-smoothing algorithm was used to smooth the surface by removing these surface artifacts.[11,12] The optimal degree of smoothing that would remove only the surface artifacts, while not altering the overall anatomical geometry, was first determined with an artificial AAA phantom.[12] By using this information, the AAA surfaces were optimally smoothed to obtain a 3D reconstruction, or "virtual AAA" (Fig 1, *C*).

Biomechanical model for abdominal aortic aneurysm wall tissue. The intrinsic mathematical relationship between stress and strain for AAA tissue needs to be characterized, based on appropriate experimental data to computationally estimate the wall stresses. Earlier studies of AAA wall biomechanics used the theory of linearized elasticity. However,

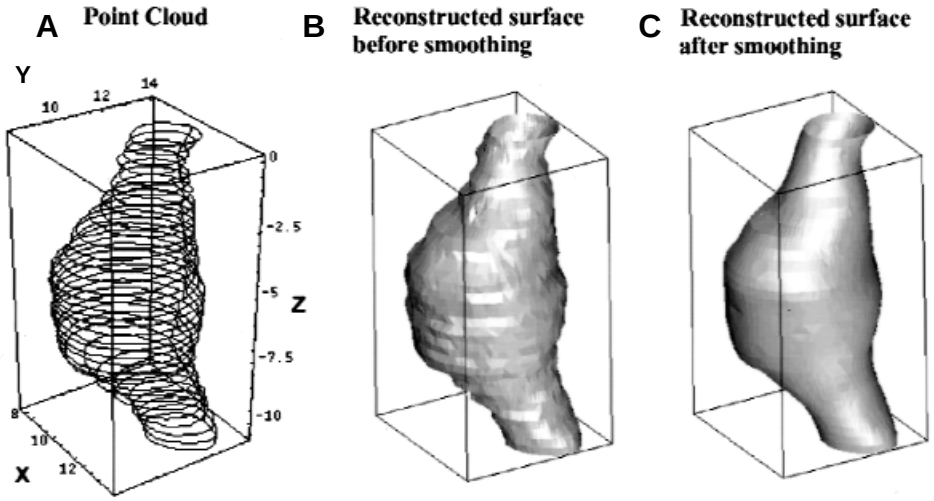

Fig 1. The abdominal aortic aneurysm point cloud (A) and the reconstructed abdominal aortic aneurysm wall surface before (B) and after (C) surface smoothing for a representative subject. The Z-axis is anatomically parallel to the long axis of the abdominal aortic aneurysm. The top edge of the abdominal aortic aneurysm represents the junction of the abdominal aortic aneurysm and the most distal renal artery branch, whereas the bottom edge represents the site of iliac bifurcation. The X and Y axes are perpendicular to the sagittal and frontal planes, respectively. Note that the surface smoothing procedure (B and C) only removed the roughness in abdominal aortic aneurysm geometry (eg, sharp corners), while preserving the overall geometric features.

this is considered largely unsuitable for biologic soft tissue in general[13,14] and AAA tissue in particular. In this study, therefore, we used a nonlinear mathematical model that was developed and validated by our laboratory, based on experimental data from 69 AAA wall specimens (see Appendix).[10,15] The mechanical properties (ie, the parameters of the mathematical model) of a typical AAA wall were determined from this experimental data and used for the present analysis (see Appendix). Previous studies in our laboratory revealed that the use of population mean values as mechanical properties for all AAA patients does not affect the results in a significant manner[10,15]; that is, the wall stresses are insensitive to variations in the values of the mechanical properties within a reasonable domain, avoiding the need for patient-specificity in AAA wall mechanical properties.

Forces and constraints on the abdominal aortic aneurysm wall. The pulsatile blood pressure within the AAA acts on its inner wall surface. Therefore, the blood pressure was measured in all subjects with a standard sphygmomanometer, both before and after CT scanning. For each subject, the systolic values of blood pressure were averaged, and the mean value was numerically applied as uniformly distributed internal forces acting outwardly on the AAA wall. The rationale behind using only the systolic pressure in a quasistatic stress analysis was that we were interested in evaluating the maximum stress acting on the AAA wall to assess its rupture risk. The shear stress induced by blood flow was neglected in this study, because it was not expected to affect the results of the stress analysis.[27] The proximal and distal ends of the virtual AAA were constrained from deforming in the longitudinal direction to simulate the tethering of the AAA at the renal artery junction and the iliac bifurcation. Residual stresses that may exist within the aortic wall in vivo and tethering forces on the posterior surface caused by the lumbar arteries were neglected.

Computational stress analysis. Because of the complex nature of the AAA geometry, the finite element method (FEM) was used as a means of solving the problem. The virtual AAA and control aortic walls were discretized into approximately 5000 quadrilateral thin-walled shell elements (creating a mesh of "finite elements") with the computer-aided design software Pro-Engineer (version 16.0, Parametric Technology Corporation, Waltham, Mass). The assumption that the AAA wall is a thin-walled shell is reasonable, because the thickness of the wall is less than 10% of the radius of curvature at all points, except perhaps those near the proximal and distal necks.[10,12,16] This mesh refinement was shown to be adequate for accurate computation of

Table I. Relevant clinical information and geometric characteristics

Subject	*Sex*	*Age (y)*	*Mean systolic pressure (mm Hg)*	*AAA diameter (cm)*	*AAA height (cm)*	*AAA volume (cm^3)*
Control	F	37	118	2.0	11.2	25.7
1	M	85	120*	6.0	10.0	143.1
2	M	86	128	6.1	9.4	155.5
3	M	77	115	5.2	10.7	120.1
4	M	77	188	5.5	10.8	145.2
5	M	73	126	6.4	16.2	271.7
6	F	74	155	6.4	10.5	216.6
Mean ± SEM†		79 ± 2	139 ± 11	5.9 ± 0.2	11.3 ± 1.0	175.4 ± 23.4

*Blood pressure was unavailable for this subject and therefore was assumed to be 120/80 mm Hg.
†Excluding the control subject.
AAA, Abdominal aortic aneurysm; *F,* female; *M,* male.

wall stresses.[10] Because no information was available about the wall thickness of each AAA, we used mean measures obtained during our earlier experimental investigations of 132 aneurysmal and 37 nonaneurysmal abdominal aortic specimens.[10] Specifically, all AAAs were assumed to have a uniform wall thickness of 1.9 mm, whereas the nonaneurysmal aortic wall was assumed to be 1.5 mm thick. A computational quasistatic stress analysis was performed on the infrarenal aorta of each study subject by using the FEM software ANSYS (version 5.3, ANSYS, Houston, Pa[17]) and the mechanical model described elsewhere[10,15] and summarized in the Appendix.

Because the assumption of constant aortic wall thickness is critical in stress analyses, we evaluated the subsequent error involved. For this, two additional stress analyses were performed on each study subject by setting the wall thickness at the lower and upper 99% confidence levels, based on available experimental data. For the control aortas (N = 37), the lower and upper 99% confidence levels were 1.28 mm and 1.78 mm, respectively. For the AAAs (N = 132), the lower and upper 99% confidence levels were 1.79 mm and 2.10 mm, respectively.

Correlation between peak wall stress and clinically measurable factors. To understand how stress is affected by simple and clinically measurable factors such as AAA diameter, height, and volume, we examined the linear correlation between peak wall stress (defined as the maximum stress found anywhere on a particular aortic wall) and these factors. The AAA diameter was defined as the maximum transverse dimension of the cross section, anywhere along the height of the AAA, and determined directly from the CT scan images. The height was defined as the straight-line distance between the centroid of the abdominal aorta at the most distal renal artery to that at the iliac bifurcation. The volume was determined by first calculating the area of each cross section and numerically integrating this data over the height of the AAA and normal aorta. Details of the numerical methodology used in these calculations are given in the Appendix.

RESULTS

The AAA geometry varied widely among study subjects. The geometric parameters and relevant clinical information for all study subjects are given in Table I.

The von Mises stress distribution on the AAA of each study subject was plotted and observed to easily represent and interpret the computational stress analysis results. "Stress" is a tensor quantity with nine components. The von Mises stress is a stress index especially suited for failure analysis and is a combination of these components. Studying von Mises stress, rather than each component of stress, allows for meaningful interpretation of the results. The 3D distributions of von Mises stress on the aortic wall of all study subjects are shown in Fig 2, *A* through *G*. Artificial stress concentrations, induced because of longitudinal constraints at the proximal and distal edges, existed in some AAAs and were much higher than the stresses in other regions of the same aneurysm. These were neglected as edge effects, and such regions are indicated on the color mapping of stresses in Fig 2. The peak wall stress (after correcting for edge effects) was recorded for each subject. Fig 3 shows the peak wall stress for each subject and the "error" caused by variation in wall thickness within its 99% confidence domain. So that the results shown in Fig 3 can be observed within the context of AAA rupture, the mean failure strength for AAA tissue, as determined from previous experimental investigation by our group, is also marked on Fig 3.

Statistical correlation of peak wall stress in the AAAs

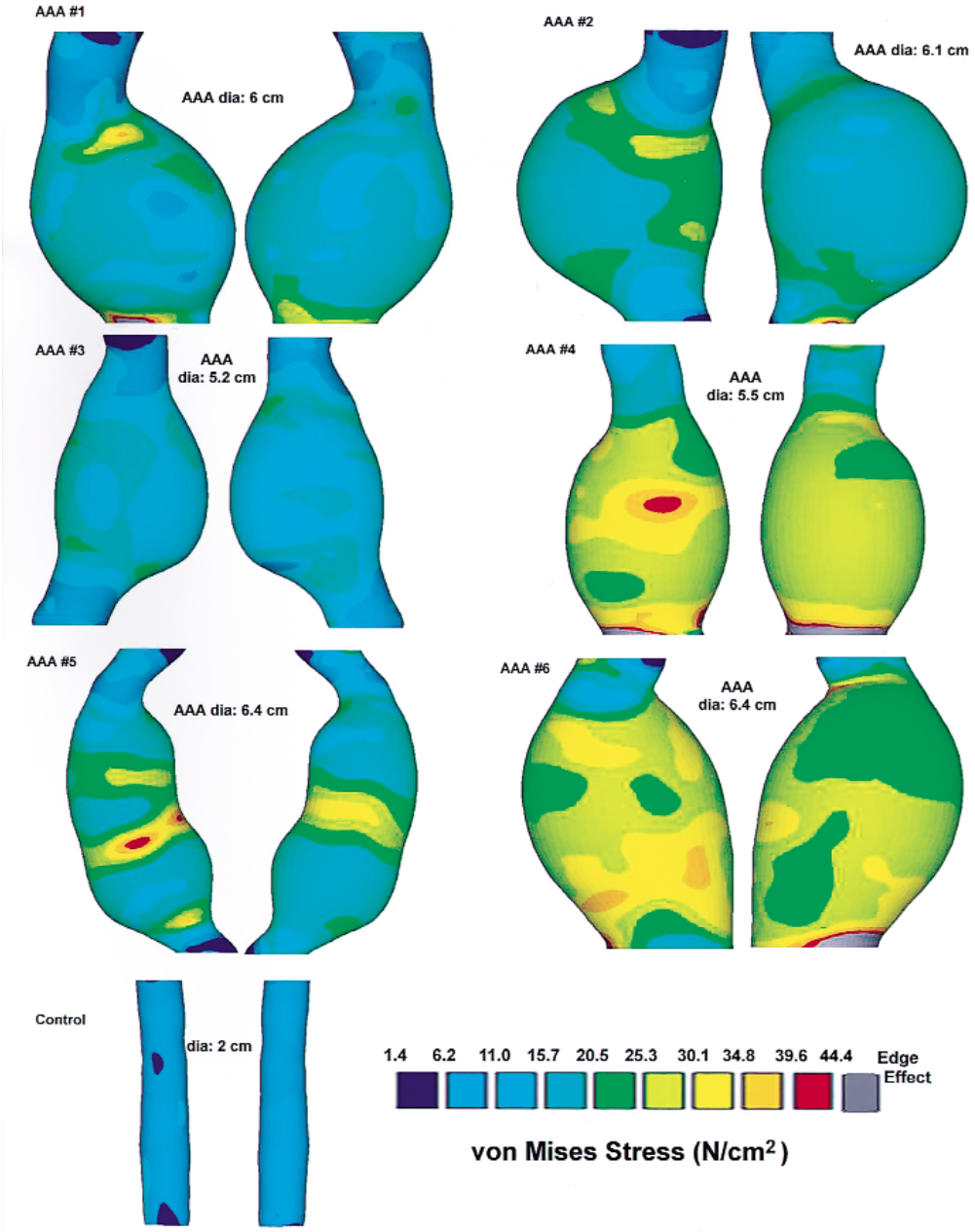

Fig 2. The distribution of von Mises stress on the posterior and anterior abdominal aortic walls of the control subject and the patients with abdominal aortic aneurysms. Grey regions are those with artificially high stress concentrations because of edge effects. The individual color scales give the stress magnitude. In all cases, blue represents the lowest stress magnitude, and red represents the highest stress magnitude. Note the comparatively lower range of stress in the control aorta. (Three-dimensional animations demonstrating these results in more detail can be found on the World Wide Web at http://www.pitt.edu/~vorp.)

with the clinically measurable factors such as height, midsection diameter, and volume is indicated in Table II. The control aorta was excluded from these calculations. Of the factors studied, AAA volume appears to have the strongest correlation with peak wall stress.

DISCUSSION

In this study, we developed and demonstrated a noninvasive methodology to estimate AAA wall stress distribution on a patient-to-patient basis. By using techniques in imaging, image processing, 3D

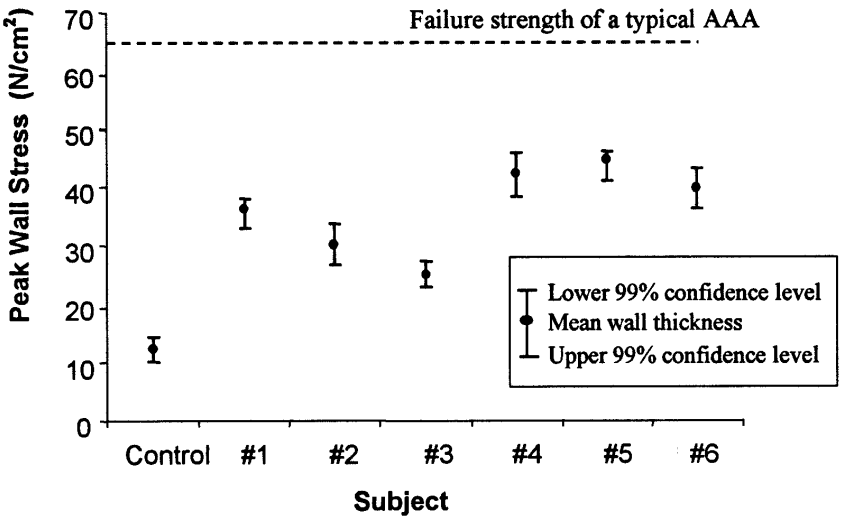

Fig 3. The peak wall stress for each study subject and the effect of wall thickness. Each subject's aortic wall was subjected to three stress analyses: with the wall thickness set equal to the population mean and the lower and the upper 99% confidence levels. The error bars indicate the range of peak wall stress for each case within that confidence domain. The *dashed line* indicates the failure strength of the abdominal aortic aneurysm, determined from unrelated experimental data,[9] and represents approximately the maximum allowable stress in a typical abdominal aortic aneurysm before rupture.

reconstruction, and engineering mechanics, we computed individually the wall stress distribution in the abdominal aorta of six patients with aneurysms and one control subject who did not have an aneurysm. Based on our results, AAA wall stress was indicated to be complexly distributed with large regional variations (Fig 2). The wall stress on the control aorta, however, was relatively low and uniformly distributed. The peak wall stress computed for the AAAs ranged from 29 N / cm^2 to 45 N / cm^2, whereas the peak wall stress computed for the control aorta was 12 N / cm^2 (Fig 3). In earlier experimental investigations,[6,7] we found that the failure strength of a typical AAA wall is 65 N / cm^2, whereas the nonaneurysmal aorta can withstand stresses as high as 121 N / cm^2. Therefore, the peak wall stress on the AAAs studied was anywhere from 45% (29:65) to 69% (45:65) of their failure strength (ie, their capacity to withstand stress), whereas the peak wall stress of the nonaneurysmal aorta was less than 10% (12:121) of its failure strength. Because AAA rupture occurs when the wall stress reaches its failure strength, such comparisons of peak wall stress to failure strength may be clinically useful in assessing rupture risk. For all the AAAs studied, the location of peak wall stress was the posterior surface. If failure strength does not vary regionally within the AAA, the site of maximum stress would also likely be the site most susceptible to rupture. Darling et al inspected autopsies of 118 patients with ruptured AAAs and found that in 82% of the cases, the rupture occurred on the posterior surface.[8]

Statistical analysis of the computed geometric factors suggests that AAA volume, rather than AAA diameter, is the best indicator of peak wall stress and, consequently, AAA rupture (Table II). Although we used a more stringent and accurate numerical integration approach in this study (see Appendix), AAA volume may be approximately measured in a clinical setting by determining the diameter at each cross section along the AAA (D_i) and using this equation:

$$\text{AAA volume} = \sum_{i=1}^{N-1} \frac{\pi s}{8} (D_i^2 + D_{i+1}^2)$$

in which D_i is AAA diameter at a given cross section i, s is the slice spacing, and N is the number of slices or cross sections that span the height of the AAA.

Despite the small patient population studied, some interesting observations were made. For example, the aneurysms of both patient 5 and patient 6 had the same midsection diameter (6.4

Table II. The correlation coefficients of clinically measurable factors with peak wall stress on the 6 AAAs.

Clinical factor	*Correlation coefficient with peak wall stress**
AAA diameter	0.56
AAA height	0.57
AAA volume	0.70
Systolic pressure	0.55

*Excluding the control.
AAA, Abdominal aortic aneurysm.

cm), but patient 5 had "normal" blood pressure (125/68 mm Hg), whereas patient 6 was hypertensive (155/90 mm Hg). According to present clinical thinking, patient 6 would be thought of as being at greater risk for AAA rupture. However, our computational analysis reveals that the peak wall stress for the AAA of patient 5 is 13% higher than that of patient 6. Based on our biomechanical perspective, the AAA of patient 5 would have a greater risk for rupture, despite having the same AAA diameter as patient 6 and lower blood pressure. The disagreement between conventional thinking and the results of the present study may be attributed to the fact that, while our analyses incorporated the effect of AAA shape (which is a critical determinant of wall stress), this is not considered in a clinical setting to assess rupture potential. We believe that this illustrates the possible clinical usefulness of an individualized biomechanical assessment of AAAs, such as that performed in this study. At the least, this would be an improvement over a generalized "rule-of-thumb" indicator of rupture (eg, the critical AAA diameter criterion).

Most earlier attempts to identify factors that influence the propensity of a given AAA to rupture have used an empirical approach. Clinical factors, such as patient age, blood pressure, AAA size, and presence or absence of thrombus, have been assessed for possible correlation with the incidence of rupture.[18-22] Although this empirical approach has helped shape the present clinical treatment of patients with AAAs, there is still substantial room for improvement. The biomechanical approach to predicting AAA rupture is fundamentally different from the earlier school of thought in that it views stress on the aneurysmal wall as the one direct factor that represents the manifestations of other critical factors, such as patient age, sex, blood pressure, AAA size, and AAA shape. Earlier studies on AAA biomechanics have mainly concentrated on comprehending the underlying problem. For example, experimental investigations of AAA wall tissue provided insight on the mechanical changes accompanying aneurysm development, while also providing sufficient data for the development of mathematical models to characterize aneurysm tissue.[6,7,23] Earlier computational investigations on wall stress in hypothetical, idealized AAAs have helped establish the factors that influence wall stresses in AAAs and have demonstrated the importance of our approach. In 1986, Stringfellow et al used the law of Laplace to determine the wall stresses in a hypothetical AAA by idealizing its geometry as cylindrical or spherical.[1] A simplified two-dimensional stress analysis was also performed to evaluate the effect of aorta-aneurysm geometry on the wall stresses. Similar two-dimensional analyses were reported by Inzoli et al, Mower et al, and Elger et al.[2-4] Our recent investigations with hypothetical, asymmetric 3D models of AAAs showed that wall stress is greatly dependent on the shape as well as the size of the aneurysm.[5] Two major assumptions inherent in all these earlier studies were the use of idealized geometry and simplified mathematical models for AAA tissue. The present study extends the earlier studies by using the actual 3D abdominal aortic wall geometry and blood pressure measured individually during CT scanning. A nonlinear mathematical model developed specifically for AAA tissue, based on previously collected experimental data, was used. Overall, this is the first attempt at estimating AAA wall stress distribution on a patient-to-patient basis, and therefore, it represents advancement over previous work in this field.

Some limitations to the present methodology exist, mainly because of limitations in contemporary imaging technology and lack of experimental information. Perhaps the most serious assumption is that of uniform and constant wall thickness. The inability to determine wall thickness reliably from CT images leads to the use of the population mean values for all subjects in the study. Fig 3 provides insight on the error involved because of this assumption. It shows that within the 99% confidence domain for wall thickness, the peak wall stress varied by plus or minus 17% from the mean for the control aorta and anywhere between plus or minus 5% to plus or minus 12% from the mean for the six AAAs. Additionally, it is unknown whether there are regional variations in wall thickness within a given AAA. Improvements in imaging technology in the future would allow for reliable, noninvasive estimation of AAA wall thickness. Another possible limita-

tion in this study is that the presence of thrombus has been ignored. Recent studies with hypothetical models suggest that intraluminal thrombus may act to reduce stress in the AAA wall, and this effect is dependent on its geometry.[24-26] Future studies should incorporate the contribution of thrombus to the mechanics of the problem. Another important assumption is that the mechanical properties do not vary regionally within a given AAA. The wall may be inherently more compliant in certain regions and stiffer in other regions. For example, if the mechanical properties of the anterior wall of the AAA are different from that of the posterior wall because of the preferential bulging, it could affect the present findings. Tissues from various regions of the same AAA need to be excised, mechanically tested, and compared to address this possibility. Another assumption in the mechanical model is that the AAA wall is isotropic (ie, mechanical properties are the same in all directions at any given point in the tissue). Our earlier finding that the mechanical properties of the circumferentially oriented AAA tissues were no different than those of the longitudinally oriented supports this assumption.[6] The shear induced by blood flow was neglected, because it contributes to only a insignificant amount of shear stress in the AAA wall (40×10^{-5} N/cm^2 at the most[27]). Possible existence of residual stresses (stresses that exist in some tissue even after all the forces are removed from it), longitudinal "tethering" forces, and posterior tethering by lumbar arteries have not been considered, mainly because no studies have evaluated these forces vis-à-vis AAAs. If a future study were to evaluate these forces, then they may be incorporated into the current computer model, simply as additional boundary conditions.[28] Because blood pressure varies periodically in vivo, a dynamic analysis would have been more realistic. The present analysis was based on a quasistatic application of systolic blood pressure, essentially because we were more interested in the peak wall stress than in the dynamic variation of stress during a cardiac cycle. We used the von Mises stress index to represent "stress" mainly to keep with convention.[2,29] A suitable failure theory for AAA tissue is presently lacking. The patterns of maximum principal stress distribution, which is another commonly used indicator of "stress" in 3D structures,[28] was almost identical to that of von Mises stress, with the former being consistently 8% higher at all points.[10] Thus, no matter which of these two quantities are used, the interpretations would not change significantly. Additionally, not all AAAs start immediately below the renal arteries or extend all the way to the iliac bifurcation, as suggested by our definition of AAA height. Indeed, some of the AAAs in this study have a proximal neck of relatively normal caliber aorta distal to the renal arteries and proximal to the start of the AAA. For this reason, a more robust definition of the AAA height should be adopted in future studies. Finally, because of the small patient population studied, the finding that AAA volume is a better indicator than diameter of peak wall stress, and consequently AAA rupture, should not be considered conclusive and/or a recommendation for clinical use.

Despite the stated limitations, this study represents a significant advancement in incorporating biomechanical principles in the clinical assessment of AAAs. Unlike earlier studies, the actual AAA geometry and blood pressure of study subjects were used for stress analysis. Also for the first time, a nonlinear material model developed solely based on data for AAA tissue was used. Besides being a completely noninvasive approach, the overall methodology required no additional involvement by the patients and is quite feasible in a clinical setting.

Future work. Before this methodology can be used clinically to aid in the management of AAAs, additional studies are necessary. In addition to improving on the stated limitations, future studies should include evaluation of changes in wall stresses with time in individual intact AAAs, with careful attention being paid to the wall stress distribution in any aneurysms that subsequently rupture. The effect of other relevant factors on wall stress, such as rate of change of blood pressure (dP/dt) could also be explored as part of a dynamic stress analysis, as opposed to the present quasistatic analysis. For wall failure strength, stochastic tables could be constructed from mechanical testing data, which correlate wall strength with various clinically measurable factors (eg, AAA size, patient age, sex, body surface area, and hypertension). This will allow for the prediction of a more patient-specific AAA wall failure strength, which may then be compared with computed wall stress distribution to assess rupture potential more accurately. Finally, we believe that this technique to noninvasively estimate regional variation of wall stresses in intact AAAs could be used as a research tool to help understand aneurysm disease itself. For example, studies may be designed to investigate the correlation of local wall stress with local expression or suppression of genes or with mural microstructure. Such studies could provide insight on the role of mechanical stresses in the natural history of AAAs.

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APPENDIX

Calculation of geometric characteristics. The height, midsection diameter, and volume of the abdominal aortic aneurysms and control aorta were calculated with the software package Mathematica (version 2.0, Wolfram Research, Champaign, Ill). First, the area and centroid of each cross section were determined by numerical integration. If X and Y Cartesian coordinate axes lie on the plane of each cross section, and the Z axis is perpendicular to the plane of the cross section, then the area of a single cross section, A[Z], is given by:

$$A[Z] = \int_{\text{all points at Z}} Y\, dX$$

The X and Y coordinates of the centroid of a single cross section ($X_c[Z]$ and $Y_c[Z]$, respectively) are given by:

$$X_c[Z] = \frac{\int_{\text{all points at Z}} X^2\, dY}{A[Z]} \quad and \quad Y_c[Z] = \frac{\int_{\text{all points at Z}} Y^2\, dX}{A[Z]}$$

The aorta height, H, was defined as the straight-line distance between the centroids of the proximal and distal cross sections of the aorta and given by the distance formula:

$$H = \sqrt{(X_c[Z_{distal}] - X_c[Z_{proximal}])^2 + (Y_c[Z_{distal}] - Y_c[Z_{proximal}])^2}$$

The aorta volume, V, was defined as the integration of the areas of cross sections over the height of the aorta:

$$V = \int_{Z_{proximal}}^{Z_{distal}} A[Z]\, dZ$$

Biomechanical model. The abdominal aortic aneurysm material was modeled as hyperelastic, homogenous, isotropic, and incompressible. The functional form of the strain energy equation, particularly derived by using mechanical testing data for abdominal aortic aneurysm tissue[10,14] was:

$$W = \alpha (I_{\underline{B}} - 3) + \beta (I_{\underline{B}} - 3)^2$$

in which W is the strain energy density, α and β are the material parameters representative of mechanical properties of the aortic tissue, and I_B is the first invariant of the left Cauchy-Green stretch tensor. The model parameters α and β were estimated for each specimen tested by fitting this equation to its stress-strain data. The statistical software Statistica (version 4.5, Statsoft, Tulsa, Okla) was used as a means of performing the nonlinear regression. The mean plus or minus SEM for α and β within the abdominal aortic aneurysm patient population was found to be 17.4 ± 1.5 N/cm^2 and 188.1 ± 37.2 N/cm^2, respectively.