INVITED COMMENTARY

Comments regarding ‘The Influence of Wall Stress on AAA Growth and Biomarkers’

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This is a complex study of 37 patients with abdominal aortic aneurysms that range from 40 to 55 mm in diameter, sizes that are normally followed closely and considered for intervention at the upper end of the diameter range. The authors examined the relationship between maximum aneurysm diameter (Dm), maximum aneurysm wall stress (Sm), aneurysm diameter growth rate and several circulating biomarkers of inflammation and/or degeneration. While Dm is the accepted AAA variable for size classification and management recommendations, the advent of computational methods for determining local wall stresses from CT scans has emerged in the past decade as a potentially powerful tool for predicting aneurysm rupture. However, Sm is only half of the AAA rupture equation. The other half is the degree of damage to the aneurysm wall due to several factors which lower wall rupture strength including atherosclerosis and deformation associated with growth. Mechanical failure or rupture occurs when arterial pressure induced wall stress exceeds the ability of the damaged wall to remain intact. While determination of aneurysm wall rupture strength in vivo is an elusive and complex problem, serum biomarker concentrations may indirectly provide an estimate of the degree of AAA wall damage. This study gives a glimpse of the relationships between these variables.

The potential value of Sm over Dm in predicting aneurysm rupture is illustrated in Figure 2. While the positive correlation of maximum stress with maximum diameter is expected and not a new finding, Figure 2 illustrates both the wide variability of Sm in aneurysms of similar Dm and the potential value of maximum stress relative to maximum diameter, that is, the ratio of Sm to Dm, or Sm/Dm. AAAs develop from near cylindrical aortas with low and relatively similar Sm/Dm values. With growth aneurysm geometries vary considerably between patients and Sm is rarely located in the plane of Dm, resulting in the wide variability of Sm/Dm. Figure 3 shows that the diameter growth rates of the one third of aneurysms with the lowest Sm/Dm values have significantly lower growth rates than that of the two thirds with higher Sm/Dm values. This means that maximum stress relative to maximum diameter (Sm/Dm) may be a valuable index for predicting AAA growth. Unfortunately circulating biomarkers were measured in only 18 of the 37 patients. However, in this subset, aneurysm growth rates correlate significantly with levels of matrix metalloproteinase-9 and both MMP-9 and CPR tended to increase with increasing values of Sm/Dm. These findings support the author’s hypothesis that AAAs in patients with high Sm/Dm values may undergo more rapid growth and wall damage than those with low Sm/Dm values. The shortcomings of this study are the small number of patients with serum biomarker analysis, the restriction of data to AAAs with diameters of 40 to 55 mm and the utilization of repeated measurement of aneurysm growth in a number of patients. Never the less, this study serves as a model for future investigations into the role of AAA wall stress, growth rate and serum biomarkers in estimating the probability of AAA rupture.