on the biomechanics or thickness of the aortic wall, or on the in vivo loading pressures. Demonstrating that the model predictions are robust under changes to these assumptions would seem to be a critical next step in developing patient-specific FEA as a risk assessment tool. Nonetheless, this study shows that finite element modeling can provide further insight into potential mechanisms of dissection as well as potentially creating dynamic imaging protocols that could aid in risk assessment outside of diameters alone.

See Article page 1365.

Commentary: Don't "stress" out. Dissection can be a thing of the past

William M. DeCampi, MD, PhD

In this issue of the *Journal*, Gomez and colleagues² used computational solid modeling to calculate maximum wall stress (MWS) in the ascending thoracic aorta in 221 patients diagnosed with ascending aortic aneurysm with trileaflet aortic valve. The authors found that MWS was associated with maximal aneurysm diameter, but relatively weakly, and concluded that the risk of rupture of smaller aneurysms (<5 cm) might be better determined using MWS.

The authors are wagering that MWS is a better predictor of rupture risk because it is more closely related to the hypothesized mechanism of rupture than is diameter. That makes sense. Afterall, it is tension in the aortic wall that will "pull it apart" if it exceeds wall strength. Of course, luminal pressure generates the tension, but the magnitude of tension depends on both the local and global geometry, and that relationship is complex. In a spherical container, wall stress is constant over the whole surface and given

Pressure is uniform in a container, but wall stress certainly isn't (used with permission).¹

CENTRAL MESSAGE

Computational techniques allow wall stress mapping of aortic aneurysms, but our inability to determine detailed wall structure and mechanics in vivo impedes our ability to predict rupture risk.

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by Laplace's law. Pressure, then, should be as good as tension in predicting container rupture. Even for a simple "hot dog"-shaped balloon, however, wall stress varies by a factor of 2 between the midportion and ends of the balloon. In a toroidal container, the meridional tension on the inner wall is greater than that on the outer wall. In a container whose surface makes an abrupt change in direction (like a flat-ended cylinder), wall stress at that turn (so-called "discontinuity stress") may be *ten or more times* the surface-averaged stress.³ In a "container" like the aorta, the wall shape is irregular, and the stress must be determined computationally, not by Laplace's law. This is what the authors accomplished. It is an important step in determining aortic wall mechanics.

What the authors did not show is that MWS predicts rupture. That requires a complex, prospective study of

patients imaged serially and followed long enough to observe some statistically requisite number of events (rupture/dissection). Even if one can conduct this challenging trial, it is not clear that the predictive power of MWS will exceed that of maximal diameter. First, it is not MWS per se, but rather the ratio of MWS to tissue strength (TS = the tension needed to tear the tissue) that more likely predicts rupture. As pointed out by the authors, TS cannot be measured in vivo. TS is also a moving target, as it likely decreases with time as elastin fragmentation progresses and as the wall thins. These biological processes also affect MWS itself because it, too, depends on wall structure, which cannot currently be modeled in vivo in a patient-specific manner. Finally, luminal pressure (systolic blood pressure) varies erratically with time by as much as a factor of 2. In the prospective study, no one will know what the blood pressure was when the event occurred. All these factors will produce variance in the model, perhaps as much as seen in the diameter versus rupture studies.

Several research groups are pursuing techniques to measure aortic wall mechanical properties (for example, echocardiographic and magnetic resonance strain imaging).⁴ In a futuristic scenario, we will be able to determine TS

by imaging the extracellular matrix of the multi-layered aortic wall (perhaps with catheter-based optical scattering techniques, molecular tag imaging, etc) in vivo. We will either apply these data to a computational model or match the image data to ex vivo specimens in which the hyperelastic behavior (including TS) has been precisely determined. We will do this serially in intervals dictated by the rate of change of MWS/TS in “at-risk” patients so that, if needed at all, a safe, reliably timed elective operation can be carried out. Dissection can be a thing of the past, but probably not in the near future.

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