Abdominal Aortic Aneurysm Risk Assessment: Biomechanics or Geometry-based Criterion?

Abstract:
The rupture of an abdominal aortic aneurysm (AAA) is believed to represent the culmination of a complex vascular mechanism partially driven by the forces exerted on the arterial wall. To prevent rupture, a diagnosed AAA is differentiated by its suitability for surgical or endovascular repair based on its maximum diameter or expansion rate measured over time during patient follow-up. At nearly all major hospitals in the U.S., subjects with aneurysms smaller than 5.5 cm (on average) are placed under clinical surveillance while those greater or equal than 5.5 cm or growing at a rate $\geq 0.5$ cm/year are recommended for elective repair. Since AAA is a largely asymptomatic condition, the screening necessary to measure growth rate over time often cannot be completed. It is a known fact, however, that basing the clinical management of this disease on expansion rate and/or maximum diameter is not a reliable measure of individual rupture risk. This is evident by the number of small aneurysms that rupture prior to reaching the critical diameter of 5.5 cm and the many more that are diagnosed at an advanced stage of expansion having exceeded the threshold size for intervention and yet did not rupture. This inability to predict accurately the individual at-risk status of an AAA has led to extensive research into other potential indicators of rupture or equivalent evaluation criteria for assessing the need for repair. Our laboratory is using a combination of image processing and numerical methods to model the geometry and the biomechanical environment of patient-specific AAAs. In this talk, I will describe our efforts in accurately modeling these aneurysms at the organ and tissue scales and the useful quantitative information we can predict from clinical images. This research represents a contribution to the ultimate goal of developing a computational tool that can be used in a clinical setting to assess the individual aneurysm rupture potential on the same day of AAA diagnosis.

Vita:
Dr. Finol received his B.E. degree in Mechanical Engineering from Universidad de Carabobo, Venezuela (1994), M.S. degree in Mechanical Engineering from University of Massachusetts Lowell (1997) and Ph.D. degree with a dual major in Mechanical and Biomedical Engineering (2002) from Carnegie Mellon University. He is currently an Associate Professor in the Department of Mechanical Engineering at University of Texas at San Antonio and previously a research faculty at Carnegie Mellon University’s Institute for Complex Engineered Systems (ICES). Dr. Finol operates the Vascular Biomechanics and Biofluids Laboratory and has research interests that include non-destructive tissue mechanics, fluid and solid mechanics modeling of blood vessels, design and optimization of intravascular medical devices, and medical image analysis.