Rupture risk evaluation in patients with abdominal aortic aneurysms (AAAs) remains a challenge in clinical practice. Aside from the accepted risk factors, including maximal aortic diameter and AAA expansion rate, no other new parameters have been accepted in recent decades as indications for surgical repair to create a more patient-specific rupture risk assessment.

A ruptured AAA is a biomechanical failure where the stress acting upon the vessel wall exceeds its strength. Biomechanical rupture risk assessment relates wall stress with wall strength to express the risk of AAA rupture. Mechanical stress in the vessel wall can be calculated by numerical methods such as finite element analysis (FEA). Wall strength can be expressed by a statistical model that includes risk factors such as female sex or hereditary predisposition. The biomechanical rupture risk assessment concept integrates geometric features with clinical parameters to calculate AAA risk factors including peak wall stress (PWS) and peak wall rupture risk index (PWRI). The AAA geometric description used by FEA models precisely calculates geometric indices including intraluminal thrombus (ILT) volume or centerline-based diameters.

AAA wall strength and wall stress computations are based on statistical assumptions. The latter refers to the elastic properties of the vessel wall, applied arterial pressure, and mechanical interaction of the AAA with surrounding body tissues, among other factors. Consequently, FEA models need to be calibrated to experimental findings from mechanical tissue testing, vessel wall histology, or CT observations.

FEA is of increasing scientific interest for AAA rupture risk assessment, as illustrated by the chronologic increase of publications listed on PubMed using the keywords “finite element” and “abdominal aortic aneurysms” (Figure 1). This article provides an overview of the current benefits and limitations of FEA for rupture risk assessment.

**EVOLUTION AND CURRENT RESEARCH**

The first FEA study of AAA, published in 1987, looked at the relationship between aneurysm size, wall thickness, and circumferential/longitudinal wall stress. Further examinations showed that the presence of an intact ILT could reduce stress in the underlying vessel wall, although these early studies did not include a biologically validated material model. Two studies evaluated the use of uniaxial tensile testing of AAA vessel wall samples.
to calibrate a previously proposed two-parameter material model. The hyperelastic model captured the isotropic and incompressible deformations at finite strains. Raghavan et al showed that varying tissue properties within 95% confidence intervals of the study sample resulted in a ≤ 4% change in computed wall stress, concluding that patient-specific properties might not necessarily improve FEA of AAA. A different and more detailed investigation concluded that as long as a non-linear wall elasticity model is used, wall stress predictions are insensitive to patient-specific properties. In a separate study, blood flow and shear stress had only marginal effects on wall stress. Accurate three-dimensional (3D) representation of the patient-specific AAA geometry and ILT proved to be crucial to wall stress estimation; however, segmenting the AAA from CT data into a 3D model is a time-consuming process that can take as long as 4 to 8 hours per patient (although this time can be decreased, as discussed later).

After a comparison between PWS and diameter to predict rupture or symptomatic AAAs, similar studies were subsequently published and the mechanical properties of ILT under static and pulsatile loads were further refined. Several studies evaluated the informative value of FEA in comparing asymptomatic, symptomatic, or ruptured AAAs. It must be stated that FEA software does not a priori differentiate between intact and ruptured aneurysms, and therefore, conclusions are limited. However, the biomechanical comparison of diameter-matched asymptomatic and intact symptomatic AAAs is clinically important. Khosla et al published a nine-study FEA meta-analysis of 204 asymptomatic intact AAAs and 144 symptomatic or ruptured AAAs. With statistical significance, the study showed that PWS was higher in symptomatic or ruptured AAAs compared to asymptomatic AAAs. Adjustments for blood pressure or AAA diameters, however, were not stringently performed in all studies.

Based on previous uniaxial tensile testing of AAA wall specimen, an FEA model to estimate wall strength based on patient sex, familial AAA history, ILT thickness, and normalized local diameter was developed. In subsequent publications, software systems that utilize FEA models, including A4clinics (Vascops GmbH) and BioPARR (The University of Western Australia), were used. The result is an estimate of PWRI, or synonymous rupture potential index: the local ratio between wall stress and wall strength (Figure 2). PWRI can then be converted into a rupture risk–equivalent diameter for clinical interpretability.

In four publications that used FEA, ruptured AAAs were compared with asymptomatic AAAs. In two of the publications, PWRI was significantly increased in the rupture group, whereas no significant difference was seen in the others. Asymptomatic AAAs were diameter-matched in three of these studies. Another approach was demonstrated in a study by Polzer and Gasser. They developed a probabilistic rupture risk index intended to consider uncertainties in material characteristics. The probabilistic rupture risk index showed a high sensitivity and specificity to distinguish ruptured AAAs from diameter-matched intact AAAs.

In a multicenter approach, Erhart et al performed FEA in patients with former asymptomatic AAA that ruptured at a later point in time. CTA rupture findings were compared with initial FEA parameters and a diameter-matched control group of patients with asymptomatic AAAs without a history of AAA rupture. It was shown that PWRI was significantly increased in the primarily asymptomatic AAAs that later ruptured. In sporadic cases, vessel wall locations with the highest PWRI in prerupture FEA were identical to later corresponding rupture site locations. Currently, FEA measurements such as PWS and PWRI are increased in symptomatic and ruptured AAAs, and they appear to be potential risk factors.

LIMITATIONS AND FUTURE IMPROVEMENTS

Limitations of the FEA method in AAA rupture risk assessment need to be considered. The process of creating a 3D AAA model is time-consuming and prone to error if not accurately performed. The time per FEA has decreased from 2 to 4 hours in the first studies to around 40 minutes with open-source software and 10 to 20 minutes with newer commercially available software.

Another point of discussion is the complexity of FEA models, which need to be realistic enough to make accurate predictions but ideally are able to solve the problem within minutes on a standard computer.
Aspects that should be further analyzed include vessel wall strength and thickness, as well as the interaction between these variables, the effects of calcifications, the influence of surrounding tissue, and AAA microstructural dynamics.\(^2\)

Man et al reported that the nonlinearity of the AAA vessel wall stress-strain relationship (ie, how the wall deforms under load) needs to be respected in order to make more accurate simulations.\(^3\)

FEA could also be applied to other vascular pathologies or implanted stent grafts. Biomechanical properties of stent grafts could be measured and implemented into finite element computation, but pressure conduction on the excluded AAA is an unknown factor. Additionally, endoleak prediction might benefit from combined computational fluid dynamics findings. The thoracic aorta has different and less investigated vessel wall properties as compared to the infrarenal aorta, and therefore, FEA computation models need to be adjusted for vessel wall elasticity if applied to the thoracic vascular pathologies. Vessel wall reconstruction and blood flow simulation are complex and computationally intensive in aortic dissections. Model reconstruction of false/true lumen, entries, and reentries are investigator dependent and prone to error, and they are based on detailed knowledge of fluid dynamics to calculate wall stress in aortic dissections.

**CONCLUSION**

Most studies on FEA for rupture risk assessment of vascular pathologies have their focus on AAA. These studies are small and retrospective with heterogeneous designs, and therefore, validity is limited. Findings need to be validated in a prospective, multicenter approach, and computation models need to be adjusted by findings from experimental research on AAA wall strength characteristics.

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