Is There a Role for Biomechanical Engineering in Helping to Elucidate the Risk Profile of the Thoracic Aorta?

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Clinical estimates of rupture and dissection risk of thoracic aortic aneurysms are based on nonsophisticated measurements of maximum diameter and growth rate. The use of aortic size alone may overlook the role that vessel heterogeneity plays in assessing the risk of catastrophic complications. Biomechanics may help provide a more nuanced approach to predict the behavior of thoracic aortic aneurysms. In this report, we review modeling studies with an emphasis on mechanical and fluid dynamics analyses. We identify open problems and highlight the future possibility of a multidisciplinary approach that includes biomechanics and imaging to evaluate the likelihood of rupture or dissection.

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Current clinical estimates of the rupture and dissection risk for the thoracic aorta are based almost solely on maximum diameter and growth rate. However, aneurysmal disease is characterized by a strong regional heterogeneity within the aortic segments. The use of aortic size alone may overlook the important influence of vessel heterogeneity. More nuanced and sophisticated metrics other than size measurement are needed to create a "fingerprint" of each patient's aorta at risk for catastrophic complications. Biomechanical modeling represents a promising approach to evaluate and predict the behavior of aortic aneurysms.

Although biomechanical modeling has been studied to evaluate wall stresses for abdominal aortic aneurysms (AAAs), publications for thoracic aortic aneurysms (TAAs) are limited. Significant differences in vascular biomechanics, atherosclerotic plaque distribution, and proteases pattern exist between AAAs and TAAs, suggesting that different approaches should be used in the modeling of these two different pathologies [1]. In the following review, we summarize modeling studies of TAAs, with emphasis on mechanical and fluid dynamics analysis and weakening of the aorta.

Material and Methods

A literature search for abstracts was performed using MEDLINE and the Cochrane Library from earliest available date to December 2014. The initial key words were: "biomechanics and thoracic aortic aneurysms/dissection," "fluid mechanics and thoracic aortic aneurysms/dissection," "mechanics and thoracic aortic aneurysms/dissection," "shear stress and thoracic aortic aneurysms/dissection," "wall stress and thoracic aortic aneurysms/dissection," and "weakening and thoracic aortic aneurysms/dissection." The literature review conformed to the preferred reporting items for systematic reviews statement standards [2] (Fig 1). The review included studies that were human studies and included patient-specific or anatomic realistic models. Studies that met the inclusion criteria are summarized in Table 1.

Biomechanics of TAAs

Issues With Current Clinical Assessment of Risk

TAAs have been called "the silent killer" because they are usually asymptomatic until catastrophic complications occur [37]. In addition, sudden death from TAAs may be misdiagnosed as myocardial infarction [38]. Nonetheless, the incidence of TAAs has risen during the past 20 years due to an improved detection rate and possibly to an actual increase in the number of cases [39].

The etiology of aneurysm formation is poorly understood. Theories include genetic and biomechanical causes such as mechanical stress [4, 5, 9, 17].

From a clinical point of view, estimates of rupture potential and dissection risk are based primarily on geometric factors and measured growth rate. These measures have been based on a single length obtained from computed tomography or magnetic resonance imaging and repeated over time. As the diameter of the aneurysm enlarges, the risk of rupture and dissection of TAAs is thought to increase in a nonlinear fashion [38]. However,

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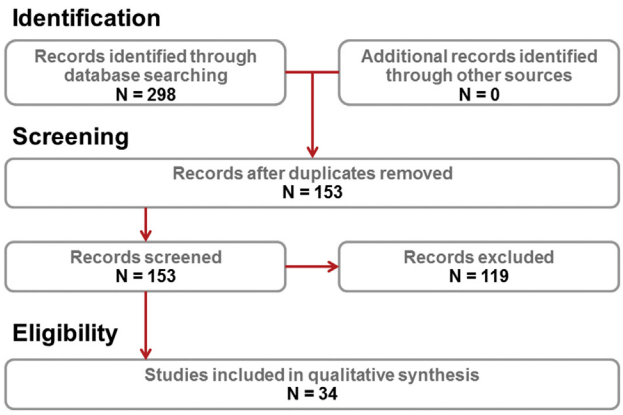

Fig 1. Four-phase flow diagram shows the number of identified articles, excluded studies, and included studies.

nearly 60% of dissections occur at a diameter not falling within current guidelines for elective surgical repair [10].

Geometry and local mechanical properties both contribute to the natural history of an aortic aneurysm. Thus, patients presenting with the same maximum diameter may have widely different outcomes due to large variations in the local degree of degeneration of the tissue. The mechanical properties of the aorta are heterogeneous [40], a consequence of the local response of the tissue to mechanical (local stress and hemodynamics) and biologic triggers (inflammation, proteases). Multilayered and asymmetric intraluminal thrombus is often present in the descending aorta and may affect the risk of rupture.

In addition to the maximum diameter, TAA growth rate has also been proposed as a predictor for aortic rupture [41]. Fast aneurysm expansion is a risk factor for TAA rupture, independently from TAA size [41]. Despite this evidence, substantial individual variation exists in aneurysm expansion rates [42], which prevents reliable prospective predictions of patient-specific enlargements.

Role of Stress in Aneurysm Rupture or Dissection

The aortic wall is a tubular structure that deforms under pulsatile blood pressure over each heartbeat. This cyclic deformation generates forces within the wall. When these forces are normalized by the cross-sectional area, they are referred to as wall stresses. Radial, circumferential, and axial stresses can be measured at every point in the vessel wall, with circumferential and axial stresses being the dominant stress components. On the intimal side, blood flowing over the arterial wall induces wall shear stress (WSS) according to the spatial velocity gradient and to the laws that govern viscous fluids (Fig 2).

Local increases in mechanical stresses result in changes in the balance of extracellular matrix production and degradation, which may result in a weaker wall [43]. Likewise, stresses caused by fluid dynamics alter genetic expression of endothelial cells [44]. This results in apoptosis and phenotypic changes that may drastically alter the structure of the vessel at the interface with flowing blood and promote thrombus formation and degradation of the underlying wall structure.

In the presence of altered geometry or altered flow patterns, or both, changes occur at the intimal layer of the aortic wall that support further wall degeneration and abnormalities in fluid dynamics (Fig 3A). Altered fluid dynamics and wall weakening may result in elevation of local mechanical stresses that affect the expression of factors that produce abnormalities in the microstructure of the aortic wall (Fig 3B). The end result is further weakening of the wall and possibly rupture of the aneurysm (Fig 3C).

Aortic rupture and dissection are both included under the same definition of acute thoracic aortic syndromes, but modeling studies suggest that the stresses that play a role in aortic dissection and aortic rupture differ. On the one hand, delamination of the layered microstructure of the aortic wall may occur if the radial wall stress exceeds the bonding forces that hold the layers together, thereby initiating dissection [25]. This may be due to a pooling of glycosaminoglycans/proteoglycans that increase interlamellar separation and initiate intralamellar delamination, leading to aortic dissection [45]. On the other hand, an aortic aneurysm ruptures when the greater of circumferential or axial stresses exceeds the failure strength of the aortic wall. Values for in vivo failure stresses have yet to be suggested for thoracic aneurysms.

Computer Simulations and Mathematical Modeling

Computer simulations and mathematical modeling enable the quantification of the biomechanical environment to which vascular tissue is exposed.

Aortic Wall Stress and Finite Element Analysis

In the clinical literature, the law of Laplace is used as a theoretic basis to define the maximum diameter as the gold standard criterion for rupture risk assessment. The law of Laplace in a tubular structure defines the circumferential wall stress (σ_{circ}) as:

$$\sigma_{circ} = \frac{PD}{2t}$$

where P is the intraluminal pressure, D is the diameter of the vessel, and t is the thickness of the wall. According to this law, the stress increases linearly with the internal diameter. This relationship is valid for elementary shapes, such as a cylinder or a sphere, that can be described with a single radius of curvature; however, aneurysms are characterized by a complex shape featuring large variability of the surface curvature [46].

For complex shapes, stresses are computed using finite element analysis (FEA) techniques. FEA allows subdividing complex geometric structures into an equivalent system of a finite number of simpler elements connected by nodes. FEA produces mathematical relationships that describe the mechanical behavior of each individual

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