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Mechanical strength of aneurysmatic and dissected human thoracic aortas at different shear loading modes

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ABSTRACT

Rupture of aneurysms and acute dissection of the thoracic aorta are life-threatening events which affect tens of thousands of people per year. The underlying mechanisms remain unclear and the aortic wall is known to lose its structural integrity, which in turn affects its mechanical response to the loading conditions. Hence, research on such aortic diseases is an important area in biomechanics. The present study investigates the mechanical properties of aneurysmatic and dissected human thoracic aortas via triaxial shear and uniaxial tensile testing with a focus on the former. In particular, ultimate stress values from triaxial shear tests in different orientations regarding the aorta's orthotropic microstructure, and from uniaxial tensile tests in radial, circumferential and longitudinal directions were determined. In total, 16 human thoracic aortas were investigated from which it is evident that the aortic media has much stronger resistance to rupture under 'out-of-plane' than under 'in-plane' shear loadings. Under different shear loadings the aortic tissues revealed anisotropic failure properties with higher ultimate shear stresses and amounts of shear in the longitudinal than in the circumferential direction. Furthermore, the aortic media decreased its tensile strength as follows: circumferential direction > longitudinal direction > radial direction. Anisotropic and nonlinear tissue properties are apparent from the experimental data. The results clearly showed interspecimen differences influenced by the anamnesis of the donors such as aortic diseases or connective tissue disorders, e.g., dissected specimens exhibited on average a markedly lower mechanical strength than aneurysmatic specimens. The rupture data based on the combination of triaxial shear and uniaxial extension testing are unique and build a good basis for developing a 3D failure criterion of diseased human thoracic aortic media. This is a step forward to more realistic modeling of mechanically induced tissue failure i.e. rupture of aneurysms or progression of aortic dissections.

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1. Introduction

Thoracic aortic aneurysms (TAAs) are localized dilatations of the ascending or descending thoracic aorta which develop over a span of years and may dissect (dissecting aneurysm) or rupture which is the most fatal condition. The mortality of thoracic aneurysms is estimated to be 50% over 5 years (Elefteriades, 2008), whereas the mortality of an untreated Type A dissection approaches 50% in the first 48 h. The pathogenesis of thoracic aneurysmal disease involves extracellular matrix degradation and loss of smooth muscle cells, causing a decrease in aortic wall integrity. The etiologies for these processes include atherosclerosis and genetic conditions such as Marfan's syndrome and Loey–Dietz syndrome (Elefteriades, 2008; Azadani et al., 2013). Hypertension has also been implicated as a cause.

Aortic dissection (AD) is an acute condition of the aorta which typically begins with a primary intimal tear on the right lateral wall of the ascending thoracic aorta (ATA), where the hydraulic shear force is at its peak, or at the descending thoracic aorta (DTA) directly after the ligamentum arteriosum (Kasper et al., 2015). The dissection first propagates in the radial direction towards the medial layer. Then, it proceeds within the media, or between the media and the adventitia, causing the layers of the aortic wall to

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separate (Mikich, 2003). The separation allows the blood flow to enter the aortic wall, whereby a secondary channel, a so-called false lumen, is created. This leads to dilatation and weakening of the remaining outer wall of the false lumen which in turn increases the probability of the rupture and causes the patient to bleed to death within minutes (Oberwalder, 2001; Criado, 2011).

Interestingly, TAAs and ADs occur at similar locations in the thoracic aorta, presumably triggered by large hemodynamic forces and tissue stresses created in the left ventricular outflow tract when the heart contracts. Furthermore, the biomechanically important constituents of the elastic arterial wall are degraded during the process of the formation of TAA and AD. The main cause of TAA or AD is assumed to be hypertension, with an occurrence of 70%, and medial degeneration of the aorta (Isselbacher, 2005; Kasper et al., 2015). Rupture of the thoracic aorta is the main reason for morbidity and mortality of patients with Marfan's or Ehlers–Danlos syndromes (Kasper et al., 2015). Due to elevated cardiovascular stress, the appearance of a dissection or aneurysm increases with gestational age, i.e. it mostly occurs in older persons (>50 years) (Oberwalder, 2001; Immer et al., 2003).

Considering the variety of reasons for developing thoracic aortic diseases, a better understanding of patient-specific biomechanical properties is essential for developing biomechanical markers to predict adverse events. Moreover, patient-specific biomechanics-based computational approaches which use wall stress and strength distributions will provide more reliable estimates of aneurysm rupture or aortic dissection initiation/progression (Vande Geest et al., 2006; Azadani et al., 2013). However, validation of biomechanics-based rupture indicators is needed before adaptation into the clinical paradigm.

A detailed analysis of the mechanical failure properties of aneurysmatic and dissected human thoracic aortas with a particular focus on four different shear tests is presented in this study. In particular, ultimate shear stresses and corresponding amount of shear values from mode II tests in four orientations, in addition to ultimate tensile stresses and corresponding stretch values from uniaxial tensile tests (in circumferential and longitudinal directions) and direct tension tests (in radial direction) of the aortic media, were determined.

2. Materials and methods

In the present study the media of diseased aortas ($n=16$; age: 58 ± 12 years) was investigated. The aortas were subdivided into three categories: 'aneurysmatic', 'aneurysmatic with connective tissue disorder (CTD)', and 'dissected'. In Table 1, the anamnesis of all donors from which the specimens were obtained are listed. Aneurysmatic specimens ($n=9$) are denoted as *AI–AIX*, aneurysmatic specimens with CTD ($n=3$) are denoted as *CI–CIII*, and dissected specimens ($n=4$) are denoted as *DI–DIV*. More specifically, the donors of the CTD specimens had fibromyxoid degeneration (*CI*), MASS syndrome (*CII*), and Marfan's syndrome (*CIII*). Fibromyxoid degeneration is the transformation of fibrous tissue into a mucous-like 'connective' tissue characterized by the accumulation of glycosaminoglycans (O'Boynick et al., 1994). Marfan's syndrome is the result of a mutation in the FBN1 gene (gene for fibrillin-1) disrupting the elastic fiber assembly in the connective tissue by altering the regulation of TGF-β production (Dietz et al., 1991; Judge and Dietz, 2005), while MASS (mitral, aortic, skin, skeletal) syndrome, also the result of a mutation in the FBN1 gene, is very similar to Marfan's syndrome but with some differences in clinical manifestations (Judge and Dietz, 2005). In addition to the anamnesis, the aortic disease and the position where the specimens were harvested are provided.

Both dissected thoracic sections and unruptured TAA sections were obtained from consented patients undergoing surgical repair at the Department of Cardiothoracic Surgery, NYU Langone Medical Center, and the Department of Cardiac Surgery, Medical University of Graz, Austria. The study protocol and the use of material from human subjects were approved by the local Ethics Committee, Medical University of Graz, Austria. In Fig. 1(a) a typically obtained aneurysmatic tissue sample (*CI*) with a severely dilated diameter is presented.

2.1. Shear testing

Tubular aortic samples were cut along the longitudinal direction to obtain flat and rectangular sheets, and the media were separated with surgical tools. With the assumption of an orthotropic structure of the aortic tissue, the behavior under six possible shear modes are identified, i.e. two different shear properties in each of the three planes (Dokos et al., 2002; Sommer et al., 2015). Using cylindrical coordinates, these planes are referred to as the $z\theta$-, rz- and $r\theta$-planes (Fig. 2). We refer to the shear modes in the $z\theta$-plane as 'in-plane' shear modes, and the shear modes regarding the rz- and $r\theta$-planes as 'out-of-plane' shear modes, and emphasize that the 'out-of-plane' shear mode should not be confused with *mode III* fracture testing. In particular, 'in-plane' shear tests in the circumferential and longitudinal directions of the $z\theta$-plane determine the ultimate shear stresses $\tau^{u}_{r\theta}$ and τ^{u}_{rz}, respectively (Fig. 2(a)), whereas shearing in the radial and longitudinal directions of the rz-plane results in the 'out-of-plane' shear stress values $\tau^{u}_{\theta r}$ and $\tau^{u}_{\theta z}$, respectively (Fig. 2(b)). In an analogous manner, 'out-of-plane' shear tests in the radial and circumferential directions of the $r\theta$-plane result in the ultimate shear stresses τ^{u}_{zr} and $\tau^{u}_{z\theta}$, respectively (Fig. 2(c)). Unfortunately, due to the restrictions arising from the specimen dimensions, we were only able to experimentally determine two out of four 'out-of-plane' shear stress values, $\tau^{u}_{\theta z}$ and $\tau^{u}_{z\theta}$.

For 'in-plane' shear tests, small rectangular tissue samples with the dimensions of 5 mm in length and 4 mm in width were prepared. An incision of 1 mm in depth along the width of the specimen was introduced to induce a predetermined breaking point, leaving the area on which the load was applied by 4×4 mm (Figs. 1(b) and 3). Representative photographs during and after a successful 'in-plane' shear test are shown in Fig. 1(c) and (d), respectively. A special specimen geometry and preparation had to be developed to ensure failure of the tissue in the correct plane during 'out-of-plane' shear tests. A variety of specimen geometries were tested to obtain the 'out-of-plane' shear stress. The final working geometry of the specimen had the dimensions 8×3 mm (length $\times$ width) with non-symmetric incisions (dashed lines) from both sides on the long edge (Fig. 4). Sandpaper and a thin consistent layer of cyanoacrylate adhesive were used to fix the specimen between two cylindrical specimen holders (Sommer et al., 2013a, 2015). Additionally, a compressive force of 0.5 N was applied to the specimens for 5 min to ensure hardening of the adhesive and proper fixation of the specimen to the specimen holders. After 5 min of adhesive hardening, the compressive force was

Table 1
Donor information such as age, gender, connective tissue disorder (CTD), and risk factor are stated. Moreover, the condition of the aorta and the harvesting position are provided.

Donor information	*Specimen denotation*															
	AI	*AII*	*AIII*	*AIV*	*AV*	*AVI*	*AVII*	*AVIII*	*AIX*	*CI*	*CII*	*CIII*	*DI*	*DII*	*DIII*	*DIV*
Institute	MUG	MUG	NYU	MUG	NYU	NYU	NYU	NYU	MUG	MUG	NYU	NYU	NYU	MUG	MUG	MUG
Age, yr	71	71	64	50	72	62	43	50	66	56	52	28	43	65	58	73
Gender	M	M	M	M	F	M	M	M	M	F	M	F	M	M	M	M
Condition	AN	AN	AN	AN	AN	AN	AN	AN	AN	AN	AN	AN	DI	DI	DI	DI
Position	ATA	ATA	ATA	ATA	ATA	ATA	ATA	ATA	ATA	ATA	ATA	DTA	DTA	ATA	ATA	ATA
CTD	–	–	–	–	–	–	–	–	–	FD	MA	MF	–	–	–	–
Risk factors	HT	HT HL	HT HL	HT HL OB	HT HM	AS	AR	AS	HT HL SM	HT SM	HT HL OB	HT	HT SM	HT	HT	HT DM

AN, aneurysmatic; AR, aortic regurgitation; AS, atherosclerosis; ATA, ascending thoracic aorta; DI, dissected; DM, diabetes mellitus; DTA, descending thoracic aorta; F, female; FD, fibromyxoid degeneration; HM, heart murmur; HL, hyperlipidemia; HT, hypertension; M, male; MA, MASS syndrome; MF, Marfan's syndrome; MUG, Medical University Graz; NYU, New York University; OB, obesity; SM, smoker.

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