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Research Paper

Layer-dependent wall properties of abdominal aortic aneurysms: Experimental study and material characterization

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ABSTRACT

Mechanical testing and in-depth characterization of the abdominal aortic aneurysm wall from fifteen patients undergoing open surgery was performed to establish the layer-dependent tissue properties that are non-available in the literature. Quantitative microscopic evaluation was performed to identify the spatial organization of collagen-fiber network. Among a number of candidate models, the four-fiber family (microstructure-motivated) model, especially that including dispersions of fiber angles about the main directions, was superior to the Fung- and Gasser-type models in the fitting quality allowed, though it presented a practical difficulty in parameter estimation, so that an analysis was conducted aiding the identification of a more specific diagonal- and circumferential-fiber family model for all three layers. The adventitia was stiffer and stronger than the other layers, owing to its increased collagen content, and its contribution to the response of the intact wall was augmented being under greater residual tension than the media, whereas the intima was under residual compression. All layers were stiffer circumferentially than longitudinally, due to preferential collagen arrangement along that axis. The histologically-guided material characterization of layered wall presented herein is expected to assist clinical decision, by developing reliable criteria to predict the rupture risk of abdominal aortic aneurysms, and optimize endovascular interventions.

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1. Introduction

Abdominal aortic aneurysm (AAA) is a general term describing the dilatation of diameter at the level of abdominal aorta to over one and a half times its normal value, owing to an underlying pathology of the aortic wall. Their presence causes concern and alertness, due to the risk of rupture leading to hemorrhage and death. They are the most prevalent aortic aneurysm type and are typically encountered in patients during the 6th or 7th decades of their life, with smoking being recognized as the most influential risk factor and hereditary predisposition considered as playing a major role (Kent et al., 2010; Lederle et al., 1997).

Proper regulation of arterial pressure with pharmacologic intervention is mandatory, so that the stresses exerted to aortic wall be reduced, but surgery is indicated when AAA diameter exceeds 5.5 cm (United Kingdom Small Aneurysm Trial Participants, 2002; Lederle et al., 2002). Nowadays, AAAs are increasingly treated with placement of an endovascular stent through the aortic lumen under x-ray control, since this provides a less-invasive approach, associated with a 3-times lower perioperative mortality rate (Greenhalgh et al., 2004; Prinssen et al., 2004). However, unlike the grafts of open surgery that are sutured to the aortic wall, the success of endovascular graft deployment and function is based on stent-tissue interaction. Excess loads by the stent might inflict injury to the aortic wall, whereas deficient loads might cause graft migration and endoleak, leading to increased rupture risk as the aneurysmal sac is submitted again to systemic pressures. Graft migration is not infrequent, with its prevalence ranging from less than 3% to 28% depending on the anatomic characteristics of AAA, the type of endograft, and the length of follow-up (Avgerinos et al., 2011).

Mechanical characterization of the AAA wall is fundamental to understanding the modes of its rupture, arising when hemodynamic stresses overcome the strength of aortic wall, and is required for the proper design of intravascular grafts and the quantitative evaluation of endovascular surgical interventions. There has, thus, been tremendous motivation to investigate the mechanical properties of AAA wall, with a plethora of notable papers published during the last three decades; reference is made to the review articles by Vorp (2007), Phillippi et al. (2011), and Humphrey and Holzapfel (2012). Still, all past studies, e.g. (He and Roach, 1994; Raghavan et al., 1996; Raghavan and Vorp, 2000; Thubrikar et al., 2001; Vallabhanemi et al., 2004; Vande Geest et al., 2006a,b; Di Martino et al., 2006; Raghavan et al., 2011; Marini et al., 2012; Forsell et al., 2013; Reeps et al., 2013; Tong et al., 2013; Tavares Monteiro et al., 2014; O'Leary et al., 2014a, 2015; Pierce et al., 2015), have considered the AAA wall tissue as intramurally homogeneous. The varying load-bearing capacity of wall layers has been presented for ascending aortic aneurysms (Sokolis et al., 2012a) and the non-dilated abdominal aorta (Weisbecker et al., 2012), but not for AAA and such data have not been included in the wall stress analysis studies hitherto performed; see the bibliography in Vorp (2007), Phillippi et al. (2011), Humphrey and Holzapfel (2012). Contrariwise, the heterogeneous nature of intraluminal thrombus present in AAA has been acknowledged by several researchers. Uniaxial and biaxial tension studies (Wang et al., 2001; Gasser et al., 2008; Tong et al., 2011, 2013; O'Leary et al., 2014b) have characterized the material response of thrombus, identifying luminal, medial, and abluminal layers.

Knowledge of the layer-dependent properties of AAA tissue is essential and has been the primary aim of this report. Particularly, we aimed at quantifying through uniaxial tensile testing with synchronous biaxial stretch recording the material and rupture properties of the intimal, medial, and adventitial layers of the AAA wall, and their residual deformations with respect to the intact wall. Phenomenological and microstructure-motivated material models are presented and compared for their efficacy as data descriptors, using histological analysis to validate our model choices and identify associations among the material response of each layer and its underlying microstructure.

Table 1 – Preoperative patient characteristics, based on clinical records, for each AAA tissue that underwent biomechanical testing.

Patient No.	Gender	Age [years]	AAA diameter [cm]	Risk factors
1	Male	66	5.5	Arterial hypertension, smoking, dyslipidemia
2	Male	76	7.0	Arterial hypertension, dyslipidemia
3	Male	85	9.0	Arterial hypertension
4	Male	73	8.5	Arterial hypertension, CABG
5	Male	80	8.5	Atrial fibrillation
6	Male	62	7.0	Arterial hypertension, smoking, CABG, PTCA
7	Male	58	5.7	Arterial hypertension, dyslipidemia, smoking, CABG, PTCA
8	Male	73	7.0	Arterial hypertension, PTCA, CABG, aortic valve replacement
9	Male	78	5.5	Arterial hypertension, dyslipidemia, smoking, CABG, PTCA
10	Male	69	15.0	Arterial hypertension, smoking
11	Male	74	4.7	Smoking
12	Male	65	7.4	Arterial hypertension, smoking, diabetes mellitus
13	Male	72	8.3	Arterial hypertension, hyperlipidemia
14	Male	69	5.9	Arterial hypertension
15	Male	71	7.1	Arterial hypertension, smoking

CABG: coronary artery bypass grafting; PTCA: Percutaneous transluminal coronary angioplasty.