Evaluating patient-specific abdominal aortic aneurysm wall stress based on flow-induced loading

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Abstract In this paper, we develop a physiologic wall stress analysis procedure by incorporating experimentally measured, non-uniform pressure loading in a patient-based finite element simulation. First, the distribution of wall pressure is measured in a patient-based lumen cast at a series of physiologically relevant steady flow rates. Then, using published equi-biaxial stress-deformation data from aneurysmal tissue samples, a nonlinear hyperelastic constitutive equation is used to describe the mechanical behavior of the aneurysm wall. The model accounts of the characteristic exponential stiffening due to the rapid engagement of nearly inextensible collagen fibers and assumes, as a first approximation, an isotropic behavior of the arterial wall. The results show a complex wall stress distribution with a localized maximum principal stress value of 660 kPa on the inner surface of the posterior surface of the aneurysm bulge, a considerably larger value than has generally been reported in calculations of wall stress under the assumption of uniform loading. This is potentially significant since the posterior wall has been suggested as a common site of rupture, and the aneurysmal tensile strength reported by other authors is of the same order of magnitude as the maximum stress value found here.

Keywords Abdominal aortic aneurysms · Non-uniform wall pressure distribution · Patient-specific analysis · Flow field measurements

1 Introduction

Abdominal aortic aneurysms (AAAs) represent permanent localized expansions of the aorta that form between the renal arteries and the iliac bifurcation. Their prevalence increases with age, and it has been estimated that they may occur in as many as 9% of males over 65 (Newman et al. 2001; Singh et al. 2001). Since AAAs form at regions in which the vessel wall is diseased and weakened, they are at risk of rupture. Johansson and Swedenborg (1986) report that mortality associated with AAA rupture can exceed 90%, accounting for 15,000 deaths annually in the U.S. alone (National Center for Health Statistics 2008, http://www.cdc.gov/nchs/deaths.htm), making AAA ruptures the 13th leading cause of death in western societies. Although AAAs can be surgically repaired by either open-abdomen or endovascular approaches if diagnosed, those procedures have their own risks. Hence clinical decisions concerning AAA management are normally made balancing the risks associated with surgical intervention against the risk of eventual lesion rupture.

Unfortunately, there does not yet exist a quantitatively accurate method for assessing failure likelihood for specific AAA patients. Current clinical practice is to evaluate the likelihood of rupture only on the basis of the maximum transverse bulge diameter, ignoring all other factors that contribute to failure. Nevitt et al. (1989) and Ashton et al. (2002) have suggested that failure probability is significantly increased when bulge diameter exceeds 5 cm. However, Darling et al. (1977) have shown that even lesions <4 cm can fail with significant
frequency. Moreover, from a biomechanical standpoint, internal stress is the physical factor that causes wall failure, not wall diameter. Accordingly, rupture can be expected to be increasingly likely as the wall stresses generated by flow-induced hemodynamic forces approach or exceed the strength of the diseased wall. In addition, local geometric factors other than diameter can be expected to alter flow field properties and lead to local stress concentrations that increase the probability of failure, while wall material property variations can alter the wall’s ability to support those stresses. Thus an accurate method for predicting risk of rupture for specific patients should properly be based on quantitative understanding of the relations between the evolution of flow fields within the lesions of those patients and the resulting wall stress development.

Although no valid technique for measuring wall stress non-invasively in vivo has been developed yet, techniques for modeling wall stress on a patient-specific basis have evolved rapidly in the last few years, representative publications being the papers by Speelman et al. (2007), Vande Geest et al. (2008), Li et al. (2008) and Rodriguez et al. (2008). Recent reviews articles are the papers by Fillinger (2006), Vorp (2007) and Humphrey and Taylor (2008). Broadly speaking, efforts to model wall stress can be categorized into two groups, (i) static and quasi-static finite element wall-only models in which no account is taken of flow within the lesion other than to assume pressure is constant along the wall inner surface and (ii) fluid–structure interaction (FSI) calculations in which a large computational package is used to compute the flow field and resulting wall stress distribution simultaneously. Efforts to perform finite element wall stress analyzes in patient-based models were introduced by Raghavan and Vorp (2000) and Raghavan et al. (2000), and have evolved rapidly in complexity from initial isotropic material models to recent studies incorporating anisotropic behavior, non-uniform thrombus distribution and wall calcifications (Raghavan and Vorp 2000; Vande Geest 2005; Vande Geest et al. 2004, 2008; Wang et al. 2002; Speelman et al. 2007). Maximum principal stress values and directions have been found to be highly dependent on the specific shape and thrombus distribution of the patient lesion and the material behavior emulated in the calculation, though values in the range of 400–750 kPa are typical. Generally consistent results have been reported by Speelman et al. (2007), Truijers et al. (2007), Vande Geest et al. (2008) and Li et al. (2008), with maximum stress values dependent on the specific model details.

A significant limitation of these methods, though, is that patient-based finite element calculations to date have relied on the assumption that the wall is subject only to uniform internal loading at constant pressure, normally a peak systolic of 16 kPa. FSI computations (Li and Kleinstreuer 2006; Papaharilaou et al. 2007; Scotti et al. 2008; Bluestein et al. 2009; Rissland et al. 2009) avoid this assumption, by calculating an intraluminal flow field and allowing non-uniform wall pressure to be elicited by the flow. However, all published FSI computations to date have assumed the flow field to be laminar. Unfortunately, it has been shown repeatedly in vivo and in vitro, in idealized and patient-based phantoms, under steady and pulsatile conditions, that AAA flow fields are usually highly non-laminar. Results published on this topic are described in the papers by Bluth et al. (1990), Asbury et al. (1995), Peattie et al. (2004, 1996a,b) and O’Rourke and McCullough (2008). Thus there remains the need for an analysis approach that calculates wall stress based on loading that replicates in vivo conditions, and incorporates the effects of wall loading when the flow is unstable.

In the present paper, we describe an initial attempt to develop a physiologic wall stress analysis procedure; that is, a finite element computational technique that evaluates the wall stress distribution and maxima in models accurately replicating the shape of individual AAA patient lesions and incorporating published wall material properties, but using load distributions derived from wall pressure measurements in corresponding experimental phantoms. As flow rate varies, the flow field may be either laminar or turbulent. In either case, the flow-induced wall pressure distribution reflects the flow field, and may or may not be stable or show nearly uniform magnitude depending on the patient geometry and flow rate. We show that the measured wall pressure distribution can be applied as loading condition for a finite element wall stress calculation, and the resulting wall stress field can be quantitatively characterized in detail.

2 Experiments

2.1 Methods

All procedures were carried out with full approval from the Institutional Review Boards of Hartford Hospital and Tufts University. The AAA models were developed from a AAA patient CT image series performed at Hartford Hospital (Hartford, CT) using a spiral CT imaging system (Model 9800 High Speed Scanner, General Electric Healthcare Inc.), with a nominal slice thickness of 0.6 mm and a helical pitch of 1.5 : 1. Images were obtained from one end of the abdomen to the other during a single sustained breath hold by the patient to minimize respiratory-induced artifacts, after intravenous administration of standard non-ionic contrast enhancement agent.

Commercial software (MIMICS V9.0, Materialise Inc.) was used to create the three-dimensional representation of the AAA lumen, which is shown in Fig. 1. Initially, the CT series contained segments of the non-dilated aorta both proximal and distal to the bulge, as well as the iliac bifurcation.