MECHANICAL WALL STRESSES AS THE POSSIBLE GENESIS OF ABDOMINAL AORTIC ANEURYSMS

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INTRODUCTION

An abdominal aortic aneurysm (AAA) is a permanent, localized dilatation of the infrarenal aorta. It is estimated that 2-3% of the population over 50 years of age have an occult AAA [1] and 200,000 new aneurysms are diagnosed each year in the United States. In most, but not all cases, the aneurysm's natural progression is that of gradual expansion. However, due to the lack of knowledge of the role that the various factors play in the enlargement process, no accurate technique exists to date to either predict the aneurysmal growth rate or the critical size at the point of rupture. Although it is widely recognized that increasing size leads to a higher risk of rupture, small size aneurysms can also rupture [2]. For lack of any other reliable method, aneurysm diameter is the current standard by which vascular surgeons estimate the risk of rupture.

The broad aim of this study is to advance the understanding of the complex interaction between the mechanical stimuli and the physiological processes responsible for the formation and enlargement of the aneurysm. One of the key possible origins of AAA is that they form secondary to changes in the mechanical stimuli, caused by architectural changes in aortic anatomy. The aim of the research is to determine the magnitude of the shear stresses acting on the aneurysm's wall and their spatial and temporal gradients at various stages of its enlargement.

METHOD

In order to understand the effect that the hemodynamic forces have on the mechanisms of formation and growth, we simulated experimentally the aneurysmal enlargement and studied the evolution of the flow inside the AAA. For this purpose, a parametric *in vitro* study of the pulsatile blood flow in symmetric and non-symmetric models of AAA has been completed, where the geometric characteristics and flow parameters of aortic aneurysms have been systematically varied.

The geometry of the symmetric models, ellipsoidal in shape, is shown on Figure 1a. We restricted the study to incipient aneurysms, before a thrombus may form in the aneurysmal cavity: the tested models have a dilatation ratio, D/d, ranging from 1.3 to 2.4 and an elongation ratio, L/d, ranging from 2.9 to 5.2. The non-symmetric models are further defined by the eccentricity of the bulge (Figure 1b).

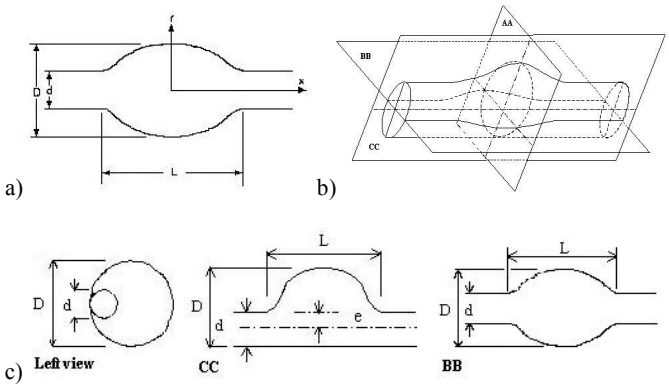

Figure 1: Plane cut of a symmetric AAA model (a), View (b) and plane cuts (c) of a non-symmetric AAA model

A piston pump (Figure 2a) reproduces the desired shape of the pulsatile flow, typically measured at the entrance of the infrarenal aorta (Figure 2b). The cardiac output was varied, simulating different cardiac conditions ranging from bradycardia, normal heart conditions to tachycardia.

The hemodynamics, inside the various models of AAA, was measured using Digital Particle Image Velocimetry (DPIV). The DPIV system is composed of two Nd:YAG lasers, a synchronizer, a CCD camera and a PC (Figure 2a) and is capable of providing quantitative, two-dimensional measurements of the instantaneous velocity vector field in a plane, with a measuring rate of 15 measurements per second. Information of the three-dimensional structure of the flow is obtained by scanning the measuring plane in successive cuts throughout the region of interest.

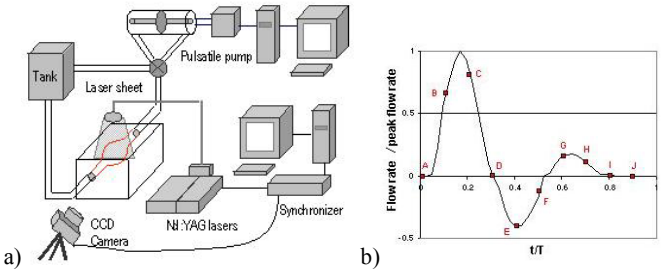

Figure 2: (a) Experimental setup, (b) Cardiac flow waveform with the measurement time locations

RESULTS

The DPIV measurements showed that, even for the case of large dilatation diameters (> 3.5 cm), the flow inside the AAA remains fully attached to the walls during systole (Figure 3-B) but massively detaches after the onset of deceleration (Figure 3-C). The flow is then dominated by the formation of regions of flow stasis with very low wall shear stresses (WSS) and the development of turbulent free shear layers.

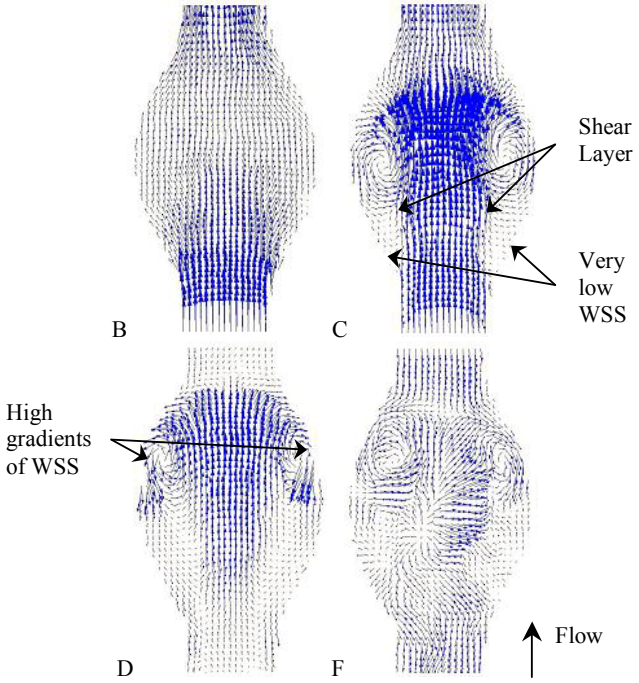

Figure 3: Flow field inside a symmetric AAA at different times in the cardiac cycle, as indicated on Figure 2b (in the case of a Womersley # of 10.7)

For a certain range of aspect ratios, cardiac rates and peak flow rates, a strong vortex ring forms, preceding the shear layer, and impinges on the distal end, generating high WSS and large spatial gradients of WSS (Figure 3-D). During diastole, the flow was found to break down inside the aneurysm into a turbulent state, which slowly dissipates during the resting period (Figure 3-F).

When the dilatation ratio is increased, carefully keeping the elongation ratio constant, an increase in the size of the recirculation region is observed. A larger portion of the proximal aneurysm wall is subjected to very low WSS, which increases the risk of particle deposit and thus of thrombus formation in the proximal half of the AAA. The magnitude of the shear stresses is found to decrease, when increasing D/d.

Increasing the elongation ratio even has a more dramatic effect on the magnitude of the WSS. Increasing L/d from 2.9 to 5.2 reduces the mean shear stresses by 50%. Furthermore, when the aneurysm is longer than the convective distance needed for the shear layer to cross the entire AAA length over one cardiac cycle, no impingement on the distal neck can further occur. Therefore, increasing the aneurysm length-wise is observed to reduce the risk of further expansion and of potential rupture. Since the WSS is the primary mechanical stimulus to which the wall reacts, it seems that the wall aims at reducing the magnitude of the experienced shear stresses by growing both radially and longitudinally.

Tachycardiac flow conditions are seen to be associated with an increase in the WSS and gradients of WSS. Increasing the mean and peak Reynolds number lead to more pathological flows.

When the AAA grows non-symmetrically, the onset of formation of flow stasis occurs at earlier stages in the enlargement of the AAA. In non-symmetric AAAs, the flow recirculates in the cavity during diastole, forming distally a stagnation point that sweeps down the bulging wall (Figures 4-E and F). Because of the presence of WSS of opposite signs, very high gradients of WSS are generated around the stagnation point.

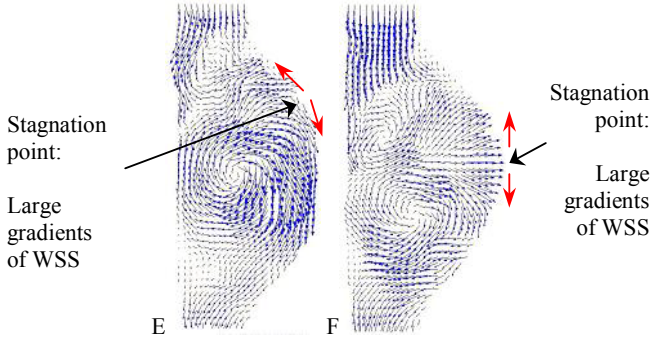

Figure 4: Diastolic flow field in non-symmetric AAA

CONCLUSION

Architectural changes in the aortic anatomy has been shown to lead to an increased area affected by stasis, as well as prolonged recirculation times associated with it. This may be the origin and cause for the progressive dilatation of AAAs. Furthermore, the appearance of distal stagnation points, with the associated region of large gradients of WSS, may be the key element to the activation of the biological processes that cause the artery to undergo changes from elastic to permanent plastic deformation.

REFERENCES

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