Haemodynamics and Stresses in Abdominal Aortic Aneurysms: A Fluid-Structure Interaction Study into the Effect of Neck and Iliac Bifurcation Angle

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ABSTRACT

Our knowledge of how geometry influences abdominal aortic aneurysm (AAA) biomechanics is still developing. Both iliac bifurcation angle and proximal neck angle could impact the haemodynamics and stresses within AAA. Recent comparisons of the morphology of ruptured and intact AAA show that AAAs with large iliac bifurcation angles are less likely to rupture than those with smaller angles. We aimed to perform fluid-structure interaction (FSI) simulations on a range of idealised AAA geometries to conclusively determine the influence of proximal neck and iliac bifurcation angle on AAA wall stress and wall shear stress (WSS).

Peak WSS and TAWSS in the AAA sac region only increased when the proximal neck angle exceeded 30°. Both peak WSS ($p < 0.0001$) and peak von Mises wall stress ($p = 0.027$) increased with increasing iliac bifurcation angle, whereas endothelial cell activation potential (ECAP) decreased with increasing iliac bifurcation angle ($p = 0.0002$) and increased with increasing neck angle.

These observations may be important as AAAs have been shown to expand, develop thrombus and rupture in areas of low WSS and here we show that AAAs with larger iliac bifurcation angles have higher WSS, potentially reducing the likelihood of rupture. Furthermore, ECAP was lower in AAA geometries with larger iliac bifurcation angles, implying less likelihood of thrombus development and wall degeneration. Therefore our findings could help explain the clinical observation of lower rupture rates associated with AAAs with large iliac bifurcation angles.

KEY TERMS

Abdominal aortic aneurysm, geometry, wall stress, wall shear stress, fluid structure interaction

INTRODUCTION

An abdominal aortic aneurysm (AAA) is a dilation of the infrarenal aorta, with a diameter at least 3cm or 1.5 times that of the normal aortic diameter. Wall stresses in the AAA have been extensively studied with regard to AAA rupture risk. These studies have indicated that peak wall stresses are significantly higher in ruptured AAAs than in both electively repaired and intact AAAs, and can be used to assess rupture risk more accurately than the maximum AAA diameter criteria (Fillinger et al., 2002; Fillinger et al., 2003; Venkatasubramaniam et al., 2004).

Aside from AAA diameter, there are several other geometric risk factors that may be important and influence the clinical management of AAA. The degree of asymmetry within the AAA has been linked with increases in wall stress, both in idealised geometries (Scotti et al., 2005; Vorp et al., 1998) and patient-specific cases (Doyle et al., 2009). Tortuosity (Georgakarakos et al., 2010) and surface curvature (Sacks et al., 1999) of the AAA are also associated with wall stress and could be useful non-invasive parameters. However, aortic proximal neck and iliac bifurcation angles are two parameters often measured in clinical practice that may influence the type of surgery (i.e. open or endovascular repair) and stent-graft device (i.e. size and type), yet have received little attention in relation to the biomechanics. Xenos et al. (Xenos et al., 2010) used patient-specific measurements from 26 AAA cases to create a range of idealised AAA geometries and then performed fluid-structure interaction (FSI) simulations to compute AAA wall stress and wall shear stress (WSS). They found non-linear relationships between von Mises wall stress and WSS with iliac bifurcation angle and aortic neck angle. In particular, their data might suggest that AAAs with larger iliac bifurcation angles are at higher risk of rupture due to the increase in von Mises wall stress, yet recent comparison of the morphology of intact and ruptured AAA indicates that AAAs with larger bifurcation angles might be less likely to rupture (Powell, 2016). Therefore, here we aim to

build upon this previous work (Xenos et al., 2010) and perform FSI simulations on similar idealised geometries in order to conclusively determine the extent that iliac bifurcation and proximal neck angle influences AAA wall stress and WSS.

METHODS

For a detailed description of the methods used, please refer to the Appendix online.

Creation of idealised geometries

We used the same geometric parameters as previously reported (Xenos et al., 2010) to create a range of idealised AAA geometries using the commercial software packages SolidWorks (Dassault Systèmes, France) and STAR-CCM+ (v11, CD-adapco, NY, USA). These geometric parameters were originally measured from a cohort of 26 AAA patients and thus represent a range of likely sizes. The neck and iliac angles were investigated in two separate parametric studies. For the iliac bifurcation angle study, the bifurcation angles were varied in 10° increments from 30° to 150°, with a constant neck angle of 0°. For the proximal neck angle study, the neck angle was varied in 10° increments from 0° to 40°, with a constant iliac angle of 60°. Figure 1a illustrates an example idealised AAA geometry with these geometric parameters.

Computational mesh, material models and boundary conditions

We used tetrahedral elements throughout the solid AAA wall (see Fig. 1b) and polyhedral elements in the fluid region (see Fig. 1c). We also used layers of prism elements at the fluid boundary and we extruded the inlet and outlets ten times the diameter. We then tested the discretisation error (study shown in Appendix). Here we opted to use a linearly elastic material model for the aortic wall (Young's modulus of 2.7 MPa and Poisson's ratio of 0.45) with a density of 2000 kg/m³ as these parameters have been used in previous FSI studies (Di

Martino et al., 2001; Leung et al., 2006; Scotti et al., 2005). Although the AAA wall is a non-linear material, the choice of material model for the wall should not significantly affect the distribution of von Mises wall stress (but will influence the magnitude) and so should not affect the relationships and overall findings of our study. Velocity and pressure waveforms were applied at the inlet and outlets of the fluid region (see Fig. 2) (Xenos et al., 2010; Olufsen et al., 2000) and a zero velocity (non-slip) boundary condition was assumed at the fluid wall, with rigid constraints at the neck and iliac boundary faces of the solid domain. As this study considers blood flow in the largest artery, we modelled blood as a Newtonian fluid with a density of 1050 kg/m^3 and a viscosity of 0.0035 Pa s . The choice of Newtonian behaviour is well justified in the large arteries as the non-Newtonian effects are only evident in smaller vessels. A mapped contact boundary interface was created between the outer surface of the fluid domain and the inner wall of the solid domain.

Data analysis

WSS, time-averaged WSS (TAWSS), oscillatory shear index (OSI) and von Mises wall stress in the AAA region were analysed for the entire range of iliac and neck angles tested. We also investigated the peak endothelial cell activation potential ($\text{ECAP} = \text{OSI/TAWSS}$) in the AAA region. We tested for correlations using the Spearman rho correlation test and deemed relationships significant when $p < 0.05$.

Figure 1: (a) Idealised AAA geometry showing the aneurysmal length, a , maximum diameter, d , iliac diameter, i , neck diameter, n , iliac angle, ϕ , and neck angle, θ . (b) Tetrahedral mesh for the solid aortic wall region showing mesh refinement at the bifurcation (insert) for the idealised AAA geometry with 0° neck and 60° iliac angle. (c) Polyhedral mesh for the fluid blood region with extrusions at the inlet and outlets (shortened for illustration purposes) for the idealised AAA geometry with 0° neck and 60° iliac angle.

Figure 2: (a) Geometry of the solid and fluid domains combined for the 0° neck and 60° iliac angle idealised geometry. The highlighted area represents the AAA region of interest where WSS and von Mises wall stress data was extracted. (b) Realistic velocity waveform for the one second cardiac cycle applied at the inlet of the fluid domain. (c) Realistic pressure waveform for the one second cardiac cycle applied at the outlets of the fluid domain. These waveforms are from Olufsen et al. (Olufsen et al., 2000) and were also used in Xenos et al. (Xenos et al., 2010)

RESULTS

Proximal neck angle and wall shear stress

As shown in Fig. 3a, we found that peak WSS in the AAA (i.e. the peak WSS observed in the AAA sac region highlighted in Fig. 2) only increased beyond a 30° neck angle (23% increase in WSS). The mean peak AAA WSS was 4.19 Pa, with maximum and minimum values of 4.99 and 3.70 Pa in the 40° and 0° neck angles, respectively. The mean TAWSS was 0.697 Pa, with maximum and minimum values of 0.793 and 0.624 Pa in the 40° and 0° neck angles, respectively (Fig.3b).

Inlet neck angle and von Mises wall stress

No obvious relationship between peak AAA wall stress and neck angle was identified (Fig. 3c) however there was a 16% increase in wall stress when the neck angle increased from 30° to 40°. The mean peak AAA wall stress was 0.203 MPa, with maximum and minimum values of 0.225 and 0.194 MPa in the 40° and 30° neck angles, respectively. Contours of wall stress at peak flow during the cardiac cycle are shown in Fig. 4a and further illustrate the relatively small changes in wall stress with varying neck angulation.

Inlet neck angle and endothelial cell activation potential

We found ECAP to increase by approximately 15% from 0° to 10° and by a further 15% when increased from 10° to 40° (see Fig. 3d). The mean peak AAA ECAP was 10.13, with maximum and minimum values of 11.67 and 8.76 in the 40° and 0° neck angles, respectively. Large regions of high ECAP were present in all of the idealised AAA geometries (see Fig. 4b), however, the size and location of these regions changed with varying neck angulation.

Figure 3: Influence of proximal neck angle on AAA sac region (a) peak WSS, (b) TAWSS, (c) peak von Mises wall stress and (d) peak ECAP. Peak WSS and peak von Mises wall stress increased when the neck angle exceeded 30° and there was an increase in ECAP at 10° and 40°.

Inlet neck angle and velocity field

As expected, varying the neck angle resulted in substantial changes in the velocity field (see Fig. 4c) as increasing the neck angle causes the symmetrical flow structure to breakdown. Once the neck is non-planar, flow tends towards the side of the AAA wall that it impacts upon entering the AAA, resulting in a large region characterised by low flow velocities on the opposite side of the AAA. However, the flow begins to depart from this side of the wall for the 30° neck angle, and then for the 40° neck angle geometry the flow moves towards the side of the wall opposite from where it initially impacts. Consequently, low flow velocities are

observed on the side of the wall initially impacted by the blood flow with these regions coinciding with locations of high ECAP.

Figure 4: AAA geometries with 0°, 10°, 20°, 30° and 40° proximal neck angle, from left to right. (a) Contours of von Mises wall stress at peak flow in the cardiac cycle. (b) Contours of ECAP averaged over the final cardiac cycle. (c) Velocity fields at peak flow.

Iliac bifurcation angle and wall shear stress

We found a strong positive relationship (Spearman $\rho = 0.98$, $p < 0.0001$) between AAA peak WSS (i.e. the peak WSS observed in the AAA sac region highlighted in Fig. 2) and iliac bifurcation angle (see Fig. 5a). Mean peak WSS across all geometries was 4.52 Pa with maximum and minimum values of 2.91 and 6.19 Pa, respectively. Therefore, peak WSS increased more than two-fold when the iliac bifurcation angle increased from 30° and 150°. There was also a strong correlation between TAWSS and iliac bifurcation angle (Spearman $\rho = 0.93$, $p < 0.0001$, see Fig. 5b). The mean TAWSS was 0.755 Pa, with maximum and minimum TAWSS of 1.0 and 0.545 Pa, respectively.

Iliac bifurcation angle and von Mises wall stress

We found iliac bifurcation angle to influence von Mises AAA wall stress (Spearman $\rho = 0.61$, $p = 0.027$, see Fig. 5c) however the relationship was not as strong as that found with WSS. The mean peak AAA wall stress for the iliac angulations was 0.207 MPa, with maximum and minimum peak AAA wall stress values of 0.243 and 0.186 MPa in the 150° and 30° iliac bifurcation angle geometries, respectively, which represents a 30% increase in peak wall stress. The distributions of von Mises wall stress at peak flow are shown in Fig. 6a.

Iliac bifurcation angle and endothelial cell activation potential

We found a strong negative relationship (Spearman $\rho = -0.86$, $p = 0.0002$) between peak ECAP and iliac bifurcation angle (see Fig. 5d). The mean peak AAA ECAP was 8.63, with maximum and minimum values of 11.41 and 7.25 in the 30° and 150° iliac bifurcation angle geometries, respectively. As shown in Fig. 6b, large regions of high ECAP (i.e. $ECAP > 8$) were present in AAAs with more acute iliac bifurcation angles, with these high ECAP regions found around the centre of the AAA where the TAWSS was lowest. As iliac angle

increased, the size of these high ECAP regions decreased rapidly and were not present in the larger iliac angulations.

Iliac bifurcation angle and velocity field

Varying the iliac bifurcation angle did not produce any distinct differences in the velocity field within the AAA. Symmetric flow was observed for all iliac angles investigated, with low velocity near the aneurysmal wall in the central region of the AAA. Figure 6c presents velocity fields at peak flow for a range of iliac bifurcation angles.

Figure 5: Influence of iliac bifurcation angle on AAA (a) peak WSS, (b) TAWSS, (c) peak von Mises wall stress and (d) peak ECAP (TAWSS/WSS). We found a positive relationship between peak WSS, mean TAWSS and peak von Mises wall stress and iliac bifurcation angle, and a negative relationship between ECAP and iliac bifurcation angle.

Figure 6: AAA geometries with 40°, 60°, 80°, 100°, 120° and 140° iliac angles, from left to right. (a) Contours of von Mises wall stress at peak flow in the cardiac cycle. The maximum difference in average wall stress was 6.5%. (b) Contours of ECAP over the final cardiac cycle. (c) Velocity fields in the AAA region at peak flow.

DISCUSSION

In this study we created a series of idealised AAA geometries and investigated the effect of proximal neck angle and iliac bifurcation angle on haemodynamics and wall stresses. We found that peak WSS in the AAA only increased when the proximal neck angle was greater than 30° , whereas peak von Mises wall stress did not increase with neck angle. Peak WSS, TAWSS and peak von Mises wall stress all significantly increased with iliac bifurcation angle, whereas ECAP decreased with increasing iliac bifurcation angle. AAAs have been shown to expand and rupture in regions of low WSS (Boyd et al., 2016; Doyle et al., 2014) and despite the increase in von Mises wall stress at larger iliac bifurcation angles, our findings could help explain why recent clinical observations report AAAs with large iliac bifurcation angles may be less likely to rupture than diameter-matched cases with smaller iliac bifurcation angles (Powell, 2016). Here we find AAAs with larger iliac angles have higher peak WSS, higher TAWSS and lower ECAP values. Although the von Mises stress increased by 30% when the iliac bifurcation angle increased from 30 to 150° , the increase in peak WSS (by 113%) and decrease in ECAP (by 57%) within the AAA region is more significant. Therefore, it is plausible that wider iliac bifurcations are protective against rupture due to the increase in WSS and decrease in ECAP in the proximal AAA sac region, which in turn decreases the likelihood of thrombus formation, wall degradation and subsequent expansion. This indicates iliac bifurcation angle could be a useful geometric variable for further investigation.

Inlet neck angle

Peak WSS occurred in the largest angle tested (i.e. 40°) and was approximately 35% higher than observed for the 0° neck angle. In previous work (Xenos et al., 2010) peak WSS in the AAA region also appears to increase with neck angle however it is difficult to determine quantitative relationships. We determined that peak von Mises wall stress in the AAA was not

strongly influenced by variations in the proximal neck angle compared to previous research suggesting that von Mises wall stress is at a maximum at 20° before decreasing slightly at 30 and 40° (Xenos et al., 2010). Although we used a different material model for the aortic wall, the stress distributions should be similar (Joldes et al., 2016) and should not affect relationships with geometry (see Appendix).

The introduction of an angled inlet neck resulted in substantial alterations in the velocity field, namely the breakdown of the symmetrical flow structure. For smaller neck angles, this asymmetric flow is characterised by high flow velocity on the side of the AAA initially impacted by the inlet flow, and low flow velocities on the opposite side. However, for larger neck angles, regions of high and low flow velocity shift and eventually move to the opposing sides of the AAA. Areas of low flow near the aneurysmal wall coincide with the regions of high ECAP. As such, the velocity fields assist in explaining why regions of high ECAP shift with increasing neck angulation.

Peak ECAP increased with neck angle and regions of high ECAP were found to occur in all of the neck angle AAA geometries tested (Fig. 4b), yet varied in location. With a straight proximal neck, a region of high ECAP is found across the whole midsection of the aneurysm where the diameter is greatest. As neck angle increases, the region of high ECAP decreases in size and shifts towards the opposite side of the aneurysm, away from the inlet. With further increases in neck angle, this region grows in size again and moves towards the opposite side of the AAA. At a 30° neck angle, the high ECAP region again circumferentially encompasses the AAA, this time towards the distal part of the sac, before finally shifting to the side of the AAA that the flow impinges on when the neck is increased to 40°. At this neck angle, high ECAP covers a large portion of the sac extending proximally and distally from the maximum diameter area. This shift in elevated ECAP is due to the asymmetry of flow within the aneurysm as the neck angle increases (Fig. 4c) and suggests that increasing neck angle, unlike increasing iliac

bifurcation angle, does not reduce the likelihood of overall growth or thrombus in the AAA, but may determine regions of local AAA expansion and thrombus development.

Iliac bifurcation angle

As the iliac bifurcation angle increased, the peak WSS in the aneurysm increased. We found significant relationships between both TAWSS (Spearman $\rho = 0.93$, $p < 0.0001$) and peak WSS (Spearman $\rho = 0.98$, $p < 0.0001$) with iliac bifurcation angle. However, the relative increase in TAWSS (27%) across the range of iliac bifurcation angles is about a quarter of the relative increase in peak WSS (113%). This suggests that the increase in WSS with iliac angulation may be amplified in localised areas of high WSS during peak flow.

Peak von Mises wall stress in the AAA significantly increased (Spearman $\rho = 0.61$, $p = 0.027$) by about 30% from the minimum to maximum iliac bifurcation angle. This is in contrast to Xenos et al. (Xenos et al., 2010) as they found more than a two-fold increase in peak AAA wall stress from 60° to 150° iliac angle.

We found a significant negative relationship between peak AAA ECAP and iliac angulation (Spearman $\rho = -0.86$, $p = 0.0002$). Regions of high ECAP were found to occur in AAA geometries with smaller iliac angles, suggesting that AAAs with smaller iliac angles may have a greater tendency towards ILT formation and wall degeneration, which could then promote expansion and rupture.

We observed little variation in velocity fields with varying iliac angle. Blood flow remained practically symmetric and was characterised by low velocities near the aneurysmal wall in the middle section of the AAA, where the diameter is largest. These sections exhibit the highest ECAP in each of the geometries due to the low TAWSS produced by these low flow velocities near the dilated AAA wall. Thus, velocity fields help to illustrate why the

regions of highest ECAP for each of the iliac bifurcation angle geometries are all located around the centre of the AAA.

Throughout this study we have attempted to compare our data to previous work (Xenos et al., 2010) and despite the different material model used, there are additional explanations for the different findings. Firstly, it is unclear if extension lengths were included into the domain of Xenos et al. Without inlet and outlet extensions in this modelling regime (similar to those shown in Fig. 2), the flow through the AAA region can be affected by the open boundaries, potentially influencing the results in the region of interest. It is also unclear if the simulation result data were independent of cardiac cycle before extracting information. In earlier work from the same group (Rissland et al., 2009) they do ensure that data was periodic after the second cardiac cycle. However it is not clear if the same approach was used in Xenos et al. As shown in Figure A1 (see Appendix), data typically becomes periodic after the third cardiac cycle and can vary significantly from the initial cycles. Furthermore the previous mesh independence study reported 6% variance in von Mises wall stress at peak systole between the final mesh and that of the prior mesh. There is also no information on the meshing approach in the near-wall boundary layer of the fluid domain which is critical for accurate WSS to be calculated. Chen et al. studied a case of aortic dissection with and without a resolved boundary layer mesh and found a 24% discrepancy in WSS (Chen et al., 2013).

Limitations

Firstly, using a FSI approach is extremely computationally intensive. In order to perform a large number of simulations and analyse the effect of the key geometric parameters (17 different geometries tested here), we made some assumptions similar to previous work (Xenos et al., 2010). We only investigated lateral iliac bifurcation angles as this angulation is more common during AAA progression. However, by assuming no anterior-posterior

angulation, the resulting stresses may be underestimated. We also limited our maximum neck angle to 40° , although in many clinical cases the neck angle could be much greater. There is also the opportunity to investigate different types of AAA to see if our findings remain valid in saccular aneurysms. We assumed the aortic wall has a constant thickness, yet in reality this wall thickness is variable. However, most importantly, we used a linearly elastic material model for the wall and so the magnitude of our wall stresses may underestimate those present in real AAAs. Nevertheless, as we note already, the findings and conclusions of this study should not be impacted by the choice of material model. Additionally, it would be useful to include idealised representations of thrombus into the geometries. This would require careful determination of where to include the thrombus and to what extent, and would create another series of idealised geometries to investigate. However, this would be useful modelling to further understand the role of biomechanics in AAA disease. Lastly, large clinical databases should be interrogated to determine the true relationship of iliac bifurcation angle and proximal neck angle with rupture rates, and to further inform clinically-relevant computational studies.

CONCLUSION

Peak WSS, TAWSS and von Mises wall stress in AAA significantly increase with iliac bifurcation angle, whereas ECAP significantly decreases. The proximal neck angle has less of an impact on AAA biomechanics. AAAs have been shown to expand and rupture in regions of low WSS (Boyd et al., 2016; Doyle et al., 2014) and clinical findings show that AAAs with large iliac bifurcation angles may be less likely to rupture than those with smaller iliac bifurcation angles (Powell, 2016). Here we show that large iliac bifurcation angles may be protective of AAA expansion and rupture by creating high WSS and low ECAP conditions.

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CONFLICT OF INTEREST

None.

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Supplementary Material

Creation of idealised geometries

All geometries have a constant aneurysmal length of 125 mm measured from the proximal neck to the base of the iliac bifurcation and have a maximum diameter of 50 mm. The diameters of the iliac arteries and proximal aorta (neck) were 10 mm and 20 mm, respectively, for all cases. As is common in AAA computational studies, we assumed a uniform wall thickness. Here we used 2 mm thickness, as implemented previously (Xenos et al., 2010). We only considered angulation in the lateral plane as this angulation is more common in human anatomy and also so we could compare our data with prior work (Xenos et al., 2010).

Governing equations

The fluid domain is governed by the Navier-Stokes and continuity equations, shown in Eqs. 1 and 2, respectively.

$$\rho \left(\frac{\partial \vec{q}}{\partial t} + (\vec{q} - \vec{q}_g) \cdot \vec{\nabla} \vec{q} \right) = -\vec{\nabla} p + \mu \vec{\nabla}^2 \vec{q} \quad (1)$$

$$\vec{\nabla} \cdot \vec{q} = 0 \quad (2)$$

Where $\vec{q}$ is the fluid velocity vector, $\vec{q}_g$ is the local coordinate velocity vector, p is the static pressure, ρ is the blood density, and μ is the dynamic viscosity. The solid domain is governed by the linear dynamics response of the system, shown in Eq. 3.

$$M\ddot{\vec{U}} + C\dot{\vec{U}} + K\vec{U} = \vec{R} \quad (3)$$

Where M is the mass matrix, C is the damping matrix, K is the stiffness matrix, $\ddot{\vec{U}}$ is the acceleration vector, $\dot{\vec{U}}$ is the velocity vector, $\vec{U}$ is the displacement vector, and $\vec{R}$ is the vector of external loads.

The FSI simulations carried out in STAR-CCM+ utilise a two-way implicit coupling between the two domains. In two-way interactions, the fluid motion and pressure affects the

structure displacement and deformation. In turn, the response of the structure affects the fluid flow. A strong implicit coupling is necessary as the simulation involves dynamic loads and velocities between a compliant structure and a relatively heavy, incompressible fluid. As a result of this coupling, data is passed between the structure solver and the fluid solver over the interface multiple times every iteration, contributing to the computational intensity of the FSI method.

Material models

Here we opted to use a linearly elastic material model for the aortic wall (Young's modulus of 2.7 MPa and Poisson's ratio of 0.45) with a density of 2000 kg/m³ as these parameters have been used in previous FSI studies (Di Martino et al., 2001; Leung et al., 2006).

Although the AAA wall is a non-linear material, at these strains the choice of material model for the wall should not significantly affect the distribution of von Mises wall stress (but will influence the magnitude) and so should not affect the relationships and overall findings of our study. We recently demonstrated that the material model does not impact wall stress for static structural simulations of AAA and that a linearly elastic model yields identical stress values as those found with a sophisticated anisotropic model and inverse solution method (Joldes et al., 2016). Additionally, several studies have shown von Mises wall stress to be practically identical whether computed using FSI or a static solid mechanics approach (Leung et al., 2006; Lin et al., 2017). Furthermore, a linearly elastic model significantly reduces the already large computational expense of FSI. Therefore, for a parametric study such as this, we believe a linearly elastic wall model is optimum. Xenos et al. (Xenos et al., 2010) employed an isotropic, hyperelastic material model (Raghavan and Vorp, 2000) and also an anisotropic material formulation (Rodriguez et al., 2008; Vande Geest et al., 2006). This anisotropic material model is significantly stiffer than the typically employed isotropic AAA material

model (Raghavan and Vorp, 2000) and yields much higher wall stresses in both AAA (Rodriguez et al., 2009) and cerebral aneurysms (Cornejo et al., 2014). However, the distribution of wall stress should be similar regardless of material model and this is shown in several studies (Joldes et al., 2016; Li et al., 2017; Polzer et al., 2013). Importantly, there is less uncertainty when using the isotropic material model as fibre orientation is not required (Lin et al., 2017; Rodriguez et al., 2009). Additionally, removing the impact of material properties is a viable pathway to clinical translation (Joldes et al., 2017).

To further demonstrate this point, we performed a series of static finite element analysis (FEA) simulations on a range of our geometries (30°, 50°, 70°, 90°, 110°, 130° and 150° iliac bifurcation angle); bearing in mind that the stress distributions between FSI and static FEA are practically identical when using the same material model (Leung et al., 2006; Lin et al., 2017) and the computational time of FSI simulations is highly expensive. We used non-linear isotropic material properties for the AAA wall (Raghavan and Vorp, 2000) and also included a simulation where the stiffness of the Raghavan and Vorp AAA material model was increased 100-fold. For these FEA simulations we used a uniform pressure boundary condition of 120 mmHg applied to the inner AAA wall and rigidly constrained the geometry at the proximal and distal regions.

We found peak AAA wall stress in these non-linear FEA simulations to be similar to von Mises stress found using our linear elastic FSI simulations in all geometries (mean difference of 5%, range 0.5 to 16%). The 30° iliac angle geometry returned the greatest difference in peak wall stress (16%) and therefore we used this geometry to compare the peak wall stress from the linear elastic (Young's modulus = 2.7 MPa) FEA simulation, using the same boundary conditions, to the wall stress obtained when using the extremely stiff (100-fold) non-linear isotropic material model. We found negligible difference (0.190 vs. 0.187 MPa). Then when we compared the data from the extremely stiff model to the peak wall stress found

using the Raghavan and Vorp material model (0.216 vs. 0.187 MPa), the difference was also 16%. This is in contrast to Xenos et al. who found peak wall stress to be 0.927 MPa in the same geometry when implementing the same material model, compared to 0.216 MPa found here. Therefore, we believe the use of a linearly elastic material model is justified in our simulations and the relationships we have observed between wall stress and geometry are practically independent of material model.

FSI simulations

We performed our simulations on two 12 core Intel Xeon CPUs with a shared memory of 64 GB RAM and used a time step of 0.001 s with a minimum of three inner iterations per time step. To minimise the simulation time, analyses were run with only the fluid mesh enabled until a steady flow pattern was established. Therefore, simulations were run as rigid wall CFD simulations until a steady cardiac cycle was observed. This process took five cardiac cycles and approximately four hours to complete. Once a steady cardiac cycle was achieved, the solid mesh and FSI solver were enabled. The CPU time for each cardiac cycle with the FSI solver enabled was around 20 hours. Therefore, the total simulation of each geometry was for six cardiac cycles and took approximately 24 hours. This solution scheme is shown in Fig. S1.

Figure S1: Sum of velocities for a line probe through the AAA region of the 0° neck and 60° iliac angle geometry over six cardiac cycles. The box on the left illustrates that a single cardiac cycle does allow sufficient time for the flow to reach a steady cyclic pattern. This is a computationally efficient method of performing FSI simulations.

Data analysis

We calculated WSS, time-averaged WSS (TAWSS), oscillatory shear index (OSI) and von Mises wall stress in the AAA region. We also investigated the peak endothelial cell activation potential ($ECAP = OSI/TAWSS$) in the AAA region with contours of ECAP produced to identify any regions that may be prone to thrombus formation and wall degeneration (Di Achille et al., 2014).

Mesh independence study

We ensured our simulations were independent of mesh size through the grid-convergence index (GCI) (Celik et al., 2008; Roache, 1994). The GCI offers a simple method for measuring the percentage difference between the value of a variable obtained between

differing mesh sizes and its asymptotic solution, where the asymptotic solution is considered to be the value obtained from an ideal mesh. We used three increasingly dense meshes generated on the 0° neck and 60° iliac angle geometry, and termed these meshes coarse, fine and finer, with the final element numbers shown in Table S1. The GCI is then calculated using Eqs. 4, 5 and 6, where ε is the relative error, f_1 is the physical parameter observed for the finer mesh, f_2 is the physical parameter observed for the fine mesh, f_3 is the physical parameter observed for the coarse mesh, p is the order of convergence, and r is the grid refinement ratio.

Table S1: Mesh details. Table shows the number of elements in each domain, number of prism layers in the fluid boundary, and the total volume mesh for the coarse, fine and finer meshes used to calculate the steady-state GCI conducted on the 0° neck and 60° iliac bifurcation angle idealised AAA geometry.

	Coarse mesh	Fine mesh	Finer mesh
Fluid domain	85549	147714	272979
Prism layers	3	6	12
Solid domain	332741	456971	733511
Total	418290	604685	1006490

$$\varepsilon = \frac{f_2 - f_1}{f_1} \quad (4)$$

$$p = \frac{\ln\left(\frac{f_3 - f_2}{f_2 - f_1}\right)}{\ln(r)} \quad (5)$$

$$GCI = \frac{|\varepsilon|}{(r^p - 1)} \quad (6)$$

The steady-state GCI was conducted with an inlet flow velocity of 0.3 m/s, which is equivalent to the peak flow during the cardiac cycle (Olufsen et al., 2000; Xenos et al., 2010). We then calculated the velocity of a line probe through the AAA and the mean WSS in the AAA region, and deemed the solution to be independent of mesh size when both these GCI values were less than 1%. The GCI values for the sum velocity of a line probe through the aneurysm and the mean WSS in the AAA were calculated to be 0.13% and 0.45%, respectively, for the fine and finer mesh. Therefore all simulations were performed using the fine mesh parameters (six layers of prism elements) with total element numbers of about 600k, depending on the geometry. The two normalised GCI parameters are shown plotted against the element count for the three volume meshes in Fig. S2 to illustrate this convergence.

Figure S2: Normalised sum velocity of a line probe through the aneurysm and the mean AAA WSS against the total number of elements in the coarse, fine and finer meshes for the steady-state GCI conducted on the 0° neck and 60° iliac angle idealised AAA geometry.

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