Blood flow and structure interactions in a stented abdominal aortic aneurysm model

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Abstract
Since the introduction of endovascular techniques in the early 1990s for the treatment of abdominal aortic aneurysms (AAAs), the insertion of an endovascular graft (EVG) into the affected artery segment has been greatly successful for a certain group of AAA patients and is continuously evolving. However, although minimally invasive endovascular aneurysm repair (EVAR) is very attractive, post-operative complications may occur. Typically, they are the result of excessive fluid–structure interaction dynamics, possibly leading to EVG migration. Considering a 3D stented AAA, a coupled fluid flow and solid mechanics solver was employed to simulate and analyze the interactive dynamics, i.e., pulsatile blood flow in the EVG lumen, pressure levels in the stagnant blood filling the AAA cavity, as well as stresses and displacements in the EVG and AAA walls. The validated numerical results show that a securely placed EVG shields the diseased AAA wall from the pulsatile blood pressure and hence keeps the maximum wall stress 20 times below the wall stress value in the non-stented AAA. The sac pressure is reduced significantly but remains non-zero and transient, caused by the complex fluid–structure interactions between luminal blood flow, EVG wall, stagnant sac blood, and aneurysm wall. The time-varying drag force on the EVG exerted by physiological blood flow is unavoidable, where for patients with severe hypertension the risk of EVG migration is very high.

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Keywords: Fluid–structure interaction; Stented abdominal aortic aneurysm; Endovascular graft; Sac pressure; Wall stress; Drag force; EVG migration

1. Introduction
Aneurysms, an irreversible ballooning of weakened artery segments, occur most frequently in the abdominal aorta. As the aneurysm expands, it may eventually rupture, making it the 13th leading cause of mortality in the US. Alternative to open surgery, where the diseased aorta segment is replaced with a synthetic graft, a minimally invasive technique has evolved over the last decade called endovascular aneurysm repair (EVAR). In EVAR, an endovascular graft (EVG) is guided from the iliac to the affected area where the EVG expands and forms a new artificial blood vessel, shielding the aneurysm from the pulsatile blood flow. The EVG or stent-graft is basically a cylindrical wire mesh embedded in synthetic graft material. While EVAR has shown outstanding success, especially for non-distorted abdominal aortic aneurysms (AAAs), EVG failure may occur due to blood leakage into the aneurysm cavity, which elevates the sac pressure and may cause rupture. This may also be caused by EVG migration when the drag force exerted on the EVG exceeds the fixation force, exposing the aneurysm sac again to the pulsatile blood flow. So far, experimental studies and computational work have focused on the analysis of blood flow induced wall stresses of either the AAA or the EVG [1–12]. For example, Fillinger et al. [1,2] computed the peak wall stresses in realistic AAA...
configurations and declared the maximum stress to be the most important indicator of AAA rupture. Di Martino et al. [12], Morris et al. [13], and Mohan et al. [14] investigated numerically or clinically the forces on a bifurcated EVG, assuming a rigid EVG wall.

Typically, the EVG is anchored to the aortic neck due to frictional forces, where arterial ingrowth and/or stent barbs and hooks may play supportive roles. Clearly, a long cylindrical AAA neck with healthy tissue is most desirable for secure EVG placement [15, 16]. Nevertheless, Resch et al. [17] reported that 45% of patients after EVAR showed migration of their EVGs. Other than problematic AAA necks, Volodos et al. [18] added systemic hypertension as a major factor in EVG migration.

So far, most publications focused on AAA-wall stress or EVG-lumen flow separately. However, a stented AAA is a complex and strongly coupled system between blood flow and EVG/AAA wall. Thus, in order to evaluate blood flow patterns, wall stress distributions, sac pressure and EVG drag force in a representative 3D asymmetric stented AAA model is shown in Fig. 1. The interacting materials include the luminal blood, EVG wall, stagnant blood in the AAA cavity, and AAA wall. The EVG is assumed to be a uniform 3D bifurcation shell attached to the proximal neck and iliac artery wall. The cavity between EVG and aneurysm wall is filled with stagnant blood, experiencing a time-dependent pressure as a result of the dynamic fluid–structure interactions. The drag force exerted on the EVG, due to fluid friction and net momentum change, was computed under physiologically realistic conditions to assess incipient EVG migration.

2. Methods

2.1. System geometry

The representative 3D asymmetric stented AAA model is shown in Fig. 1. The interacting materials include the luminal blood, EVG wall, stagnant blood in the AAA cavity, and AAA wall. The EVG is assumed to be a uniform 3D bifurcating shell attached to the proximal neck and iliac artery wall. The cavity between EVG and aneurysm wall is filled with stagnant blood, experiencing a time-dependent pressure as a result of the dynamic fluid–structure interactions. The drag force exerted on the EVG, due to fluid friction and net momentum change, was computed under physiologically realistic conditions to assess incipient EVG migration.

2.2. Flow equations

For transient three-dimensional incompressible fluid flow, the governing equations in tensor (or comma) notation, following Einstein’s repeated index convention, are:

continuity : \( u_{i,i} = 0 \) \( \quad (1) \)

momentum : \( \rho \frac{\partial u_j}{\partial t} + \rho u_j u_{i,j} = -p_{i,j} + \tau_{ij,j} + \rho f_i \) \( \quad (2a) \)

stress tensor : \( \tau_{ij} = \eta \dot{\gamma}_{ij} \) \( \quad (2b) \)

non-Newtonian fluid model : \( \eta = \frac{\eta_p}{1 + (k_1\dot{\gamma})^2(\dot{\gamma}/\dot{\gamma}_c)^2} \) \( \quad (2c) \)

where \( u_i \) is the velocity vector, \( p_i \) the pressure scalar, \( \rho \) the fluid density, \( f_i \) the body force at time \( t \) per unit mass, \( \dot{\gamma}_i \) the wall displacement velocity at time \( t \), \( ^{5}Q(t) \) the moving spatial domain upon which the fluid is described, \( \dot{\gamma}_{ij} \) the shear rate tensor, \( \eta_p \) the plasma viscosity, \( \dot{\gamma}_c \) a relative shear rate, \( \dot{\gamma}_r \) is defined by a “phenomenological kinetic model” [19], \( k_0 \) the lower limit Quemada viscosity constant, \( k_w \) the upper limit Quemada viscosity constant, and \( H_t \) is the hematocrit. The parameter values for the blood and the structures are given in Section 2.4.

2.3. Structure equations

The general governing equations for structure dynamics are:

momentum : \( \sigma_{ij} = \sigma_{ij} + \rho f_i \) \( \quad (3a) \)

\( \dot{u}_i = \frac{\partial \sigma_{ij}}{\partial x_j} \) \( \quad (3b) \)

where equilibrium of condition : \( \sigma_{ij} n_i = T_i \) on \( ^{5}\Gamma(t) \) \( \quad (4) \)

and constitutive : \( \sigma_{ij} = D_{ijkl}\ddot{x}_{kl} \) \( \quad (5) \)

Here, \( ^{5}\Omega(t) \) is the structure domain at time \( t \), \( \ddot{x}_{ij} \) the outward pointing normal on the wall surface \( ^{5}\Gamma(t) \), \( T_i \) the surface traction vector at time \( t \), \( ^{5}\Gamma(t) \) the boundary of the structure domain, \( \sigma_{ij} \) the mechanical stress tensor, \( D_{ijkl} \) the Lagrangian elasticity tensor, and \( c_{ij} \) is the strain tensor.

In order to analyze the stress distributions in the aneurysm wall, the Von Mises stress, used as a material fracture criterion in complicated geometries, is employed. Especially,

\( \sigma_{\text{Von Mises}} = \sqrt{\frac{3}{2}(\sigma_1 - \sigma_2)^2 + (\sigma_2 - \sigma_3)^2 + (\sigma_3 - \sigma_1)^2} \) \( \quad (6) \)

where \( \sigma_1, \sigma_2, \) and \( \sigma_3 \) are the three principal stresses.
Fig. 1. (a) Representative AAA with EVG, (b) system inlet and outlet profiles, and (c) actual EVG photograph (Zenith, Cook Incorporated 2003C).
Table 1: Assumptions for blood flow and structure characteristics

<table>
<thead>
<tr>
<th>Blood flow</th>
<th>Structure characteristics (artery wall, aneurysm wall, and EVG)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incompressible</td>
<td>Isotropic and elastic</td>
</tr>
<tr>
<td>Non-Newtonian fluid (Quemada model)</td>
<td>Incompressible</td>
</tr>
<tr>
<td>Laminar blood flow</td>
<td>Non-linear (large deformations)</td>
</tr>
<tr>
<td>No slip at the wall</td>
<td>No tissue growth on walls</td>
</tr>
<tr>
<td>Blood-particle effects not considered</td>
<td>No residual stresses</td>
</tr>
<tr>
<td>No endoleaks in stented aneurysm models</td>
<td>EVG consists of equivalently uniform material</td>
</tr>
<tr>
<td>Stagnant blood in cavity</td>
<td>No EVG migration</td>
</tr>
</tbody>
</table>

2.4. Numerical method

The underlying assumptions for simulating the coupled fluid–structure interactions are listed in Table 1. The blood parameter values in Eq. (2c) are \( \rho = 1.050 \text{ g/cm}^3 \), \( k_0 = 4.58619 \), \( k_\infty = 1.29173 \), \( H_l = 40\% \), and \( \eta_p = 0.014 \text{ dyn/cm}^2 \) [19]. Table 2 lists the structure parameter values used in the present simulations. With respect to Young’s moduli for the arterial wall and aneurysm, experimental data indicate that Young’s modulus of an aneurysm is much higher than for a normal artery [7]. In this paper, the Young’s modulus is assumed to be 4.66 MPa. The healthy artery section (neck) is incompressible with a Poisson ratio of 0.49, and the aneurysm wall is nearly incompressible with a Poisson ratio of 0.45 [3]. For a bifurcated NiTi-stent interwoven with graft material (EVG), no direct experimental data are available; thus, an equivalent Young’s modulus for the uniform EVG configuration was assumed to be10 MPa [20].

The physiologically representative inflow velocity waveform is shown in Fig. 1 with a maximum Reynolds number \( (Re_{\text{max}}) \) of 1950 and average Reynolds number \( (Re_{\text{average}}) \) of 330. For the outlet pressure (see Fig. 1), the peak and average pressures are 122 and 98.7 mmHg, respectively [21]. The pulse period is chosen as \( T = 1.2 \text{s} \). The inlet velocity profile is assumed to be parabolic. For the composite structures, the boundary conditions are a fixed degrees-of-freedom (DOF) at the inlet and the exit, and a free DOF on the wall. The fluid–structure interactions occur on the interfaces between luminal blood flow and EVG wall, as well as the sac’s stagnant blood and aneurysm/EVG wall.

The finite element software package (ANSYS 7.1) for linear and non-linear multi-physics analysis in Arbitrary Lagrangian–Eulerian (ALE) formulation has been employed to solve this fluid–structure interaction problem. Specifically, it uses separately ANSYS FLOTTRAN for the fluid domain and ANSYS structural solver for the solid parts. It transfers fluid forces, solid displacements, and velocities across the fluid–solid interface (see Fig. 2). A total of 76,730 8-node fluid elements were needed for meshing the luminal blood flow region, 66,820 structure elements for the EVG and AAA.
Table 3a
Comparison of results between simulations and theoretical analyses

<table>
<thead>
<tr>
<th>Straight artery</th>
<th>ANSYS FSI simulation results</th>
<th>Theoretical results [22]</th>
<th>Error (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radial deformation (internal wall)</td>
<td>0.0195 cm</td>
<td>0.0203 cm</td>
<td>4.1</td>
</tr>
<tr>
<td>Circumferential stress (internal wall)</td>
<td>0.121 MPa</td>
<td>0.115 MPa</td>
<td>5.0</td>
</tr>
<tr>
<td>Radial strain (internal wall)</td>
<td>0.012</td>
<td>0.0103</td>
<td>4.2</td>
</tr>
<tr>
<td>Radial strain (external wall)</td>
<td>0.016</td>
<td>0.0155</td>
<td>4.3</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>Maximum circumferential stress</td>
<td>0.175 MPa</td>
<td>0.16 MPa (Laplace's equation)</td>
</tr>
</tbody>
</table>

Table 3b
Comparison of results between simulations and clinical observations

<table>
<thead>
<tr>
<th>ANSYS FSI simulation results</th>
<th>Clinical observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Von Mises stress drop</td>
<td>82%</td>
</tr>
<tr>
<td>Diameter decrease</td>
<td>12%</td>
</tr>
<tr>
<td>Maximum wall displacement</td>
<td>1.52 mm in non-stented aneurysm; 0.17 mm in stented aneurysm</td>
</tr>
</tbody>
</table>

The algorithm continues to loop through the solid and fluid analyses until convergence is reached for each time step (see Fig. 2). Convergence in the stagger loop is based on the quantities being transferred at the fluid–solid interface. A variable time step was employed, where \( \Delta t = 0.005 \) s with 60 total time steps per cycle. Six cycles were required to achieve convergence for the transient analysis. Using a single processor, the total CPU time was about 50 h on an IBM p690 workstation.

2.5. Model validations

2.5.1. Comparison to theoretical results and clinical observations

In order to test the accuracy of the ANSYS-ALE-FSI solver for simulating interacting flow and wall phenomena, several computer model validation studies were performed. The comparison between our present simulations and theoretical analyses as well as clinical observations is listed in Tables 3a and 3b.

2.5.2. Comparison with Womersley's wave propagation theory

The wave propagation equation for viscous blood flow in a longitudinally tethered elastic vessel is

\[
\omega^* = c_0 \sqrt{1 - \frac{F_{10}}{1 - \sigma^2}}
\]

(7)

where \( c_0 \) is the Moens–Korteweg wave speed, which depends on the system’s geometric and material properties; \( F_{10} \) a function of the Womersley number, Bessel functions, and Young’s modulus; and \( \sigma \) is Poisson’s ratio of the elastic tube. Using a Womersley number of 10 and Young’s modulus of 1.0 MPa, compared with the wave propagation value of \( \omega^* = 831.0 \text{ cm/s} \) from Eq. (7) for a longitudinally tethered elastic tube, ANSYS-ALE-FSI simulation results yielded \( \omega^* = 880 \text{ cm/s} \), i.e., the error is 5.9%.

2.5.3. Comparison with experimental data

Gawenda et al. [26] measured the sac pressure with in vitro stented aneurysm models consisting of 6 or 12 layers of latex. The model parameters are listed in Table 3c.

Now, the sac pressures were computed for similar stented aneurysm models and flow conditions employing the ANSYS-ALE-FSI-solver. Fig. 3 presents the comparison between the experimental and numerical results. It can be seen that the simulation results are in good agreement with the experimental data.

3. Results

The results are divided into three groups, i.e., the beneficial impact of EVG insertion (Figs. 4 and 5), the luminal blood velocity fields, wall stress distributions and sac pressure levels at three selected time levels during the cardiac cycle (Figs. 6–8), and the effect of blood pressure waveforms on the transient EVG drag force, calculated from the wall
shear stress distribution and the net momentum change (see Fig. 9).

3.1. EVG impact

A securely placed EVG forms a new smooth conduit for blood flow (Fig. 1) and hence protects the weakened aneurysm wall from high pressure and stress levels (Fig. 4). In fact, in case of zero leakage into the cavity, the sac pressure is reduced by a factor of 10 and the maximum wall stress decreases by a factor of 20 throughout the cardiac cycle when an EVG is inserted (see Fig. 4a and b). As a result, the largest change in maximum AAA diameter drops during systole from 2 to 0.17 mm (Fig. 4c). Indeed, as reported by Malina et al. [24], EVG placement can reduce the maximum wall deformation to 0.2 mm.

Clearly, these dynamic system parameter variations due to cyclic fluid–structure interactions are powered by the given blood pressure waveform (cf. Figs. 1 and 4, keeping in mind that the inlet pressure differs only by 5% from the outlet pressure).

A snapshot during the critical deceleration phase ($t/T = 0.27$, peak blood pressure) shows the velocity fields as well as the pressure and wall stress distributions in both the open AAA and the stented AAA (Fig. 5). Referring to Fig. 5a, the angulated AAA neck guides the jet-like blood stream into the large cavity, forming immediately two strong vortices due to the sudden area expansion. The greatest impact of the angled blood stream occurs, as can be expected, not where the aneurysm has its maximum diameter, but well below near the iliac bifurcation, i.e., the impact area of highest net momentum change. Interestingly, the maximum wall stress and wall deflection in non-stented AAA have their highest magnitudes in different locations, i.e., the maximum wall stress (0.59 MPa) occurs on the right side above the bifurcation; while the maximum wall deformation (2.3 mm) occurs on the left side near the angled neck. Now, with an EVG in place, the blood flow is tubular and rather uniform, except for a few areas of secondary flows caused by neck and iliac angles (Fig. 5b). As a result of complex fluid–structure interactions between lumen blood, EVG wall, stagnant sac, and AAA wall, the sac pressure still remains at 14.38 mmHg, i.e., 11.8% of the lumen pressure. Triggered by the EVG wall shear stress and net momentum change, the drag force acting on the EVG is at that moment almost 2N. While the stress in the AAA wall and the pressure in the cavity are very low, the EVG carries the blood flow impact as numerically indicated with a peak EVG wall stress of 1.7 MPa at the stagnation (or bifurcation) point (see Fig. 5b and c).

3.2. Transient fluid–structure interactions

In order to illustrate the dynamics of pulsatile blood flow influencing the stented AAA parameters (see Figs. 6–8), three representative time levels were selected, i.e., reverse flow at $t/T = 0.1$ ($Re = 70$), peak systole at $t/T = 0.2$ ($Re = 1950$), and flow deceleration at $t/T = 0.27$ ($Re = 1200$). In general, the sac pressure stays very low throughout the cycle; however, it increases slightly as the flow input waveform progresses (and time elapses) because of the blood pressure transmission from the EVG lumen via the distensible EVG wall into the aneurysm cavity. The same can be observed for the peak $\sigma_{max}$ in the AAA wall, occurring always in the same location, which is basically an inflection point in the geometric mid-plane wall function. Naturally, the stress in the (healthy) neck tissue is the highest because of the necessary oversizing to keep the EVG anchored. The luminal blood flow meanders from the AAA neck part through the aneurysm with typically four small recirculation zones until it bifurcates into the EVG daughter tubes, i.e., the iliac legs. As expected, the maximum EVG wall stress can be found at the EVG bifurcation, nonlinearly increasing during the observation time. Although the present AAA geometry is anterior–posterior symmetric, the flow field and wall stress distributions are asymmetric, i.e., the results are fully three-dimensional (see Figs. 6–8).

Comparing slices C–C in Figs. 6–8 more closely, reveals that the location of the maximum EVG stress switches in the daughter tubes between up-flow (Fig. 6) and peak down-flow (Fig. 7), and back again during decelerating down-flow (Fig. 8). Strong secondary flows appear before the EVG flow...
division (see slices B–B in Figs. 6–8), an area which also experiences a relatively high wall stress.

3.3. EVG migration

If the actual EVG drag force starts to exceed the fixation force, the EVG will migrate or dislodge. As mentioned, the drag force is composed of the integral over the surface shear plus the net pressure, where the shear stress contribution is typically only 3% of the total. The fixation force for an EVG without barbs and hooks is the friction between the proximal EVG segment and the AAA neck which is usually oversized to supply, at least initially, solid anchoring. In addition, the EVG ends may be secured via frictional effects.

Fig. 4. EVG impacts on AAA: (a) EVG impact on sac pressure, (b) EVG impact on maximum wall stress, and (c) EVG impact on \( d_{\text{AAA, max}} \) changes.
Fig. 5. Comparison between non-stented AAA and stented AAA ($t/T = 0.27$): (a) non-stented AAA, (b) stented AAA, and (c) wall stress distribution in EVG.
Fig. 6. Fluid–structure interactions of stented AAA ($t/T = 0.1$).
Fig. 7. Fluid–structure interactions of stented AAA ($t/T = 0.2$).
Fig. 8. Fluid–structure interactions of stented AAA ($\tau/T=0.27$).
to the iliac lumen. In case the EVG migrates, blood can leak into the AAA cavity, leading to a dangerous pressure buildup and subsequently aneurysm rupture. As alluded to in Section 1, ideally the AAA neck should be long, cylindrical, and of healthy tissue. Obviously, AAA patients with severe hypertension (see waveform IV in Fig. 9) are especially at risk for EVG failure because in that case the drag force has a maximum and the relatively large pressure difference during the cardiac cycle may reduce the EVG fixation to a calcified, i.e., hardened, neck tissue. Specifically, the pressure descriptions of waveforms I–IV range from “normal” to “severe”, where $\Delta p_{\text{max,normal}} = 40 \text{ mmHg}$ and $\Delta p_{\text{max,severe}} = 70 \text{ mmHg}$. For example, Mohan et al. [14] declared that high blood pressure is one important factor to cause EVG migration, while Morris et al. [13] found that the drag force may vary over the cardiac cycle between 3.9 and 5.5 N. Thus, for an EVAR patient with severe hypertension, extra fixation should be considered.

Because of the incompressible-fluid condition, the transient drag force exhibits, with a very small time lag, basically the same trend as the given inlet pressure waveform (see Fig. 9).

### 4. Discussion

Although minimally invasive endovascular aneurysm repair is very attractive, post-operative complications may occur, which are the result of excessive fluid–structure interaction dynamics. Thus, the fluid–structure interactions for a representative AAA model with and without a realistic EVG have been investigated in terms of pulsatile blood flow influencing EVG movement, which is transmitted via the stagnant blood in the cavity to the aneurysm wall. Of interest are the beneficial impact of an EVG, the blood velocity field, the highest pressure level in the aneurysm sac, the stress distributions, and displacements of both of the EVG wall and the AAA wall, as well as the maximum drag force exerted on the EVG. It was readily demonstrated that a securely placed EVG shields the diseased AAA wall from the pulsatile blood pressure and hence keeps the maximum wall stress 20 times below the wall stress value in the non-stented AAA.

An interesting finding is that the sac pressure is reduced significantly but is not zero after the sac is completely excluded by the EVG; i.e., no blood leakage exists. Thus, our simulation shows that even in the absence of endoleaks, the sac pressure can be generated by the complex fluid–structure interactions between luminal blood flow, EVG wall, stagnant sac blood, and aneurysm wall. The EVG/AAA wall compliance plays an important role in sac pressure generation.

As indicated in Section 2.5, our simulation results are in good agreement with the experimental data of Gawenda et al. [26]. As shown in Figs. 7–9, the intra-sac pressure varies from 9.8 to 14.4 mmHg (4.6 mmHg pulsatility) during one cycle, while the average EVG lumen blood pressure ($p_{\text{lumen}}$) ranges from 82.3 to 120.3 mmHg (38 mmHg pulsatility). Sonesson et al. [25] found that the sac pressure pulsatility varied from 0 to 6 mmHg clinically. Dias et al. [27] indicated that the mean sac pressure pulsatility ranged from 2 to 10 mmHg for their 30 patients. Compared to the lumen pressure pulse, the pressure pulsatility in the sac is not substantial. While the AAA volume should typically shrink after successful EVAR, aneurysm enlargement might still occur because an elevated sac pressure could cause a delayed aortic aneurysm enlargement, even after successful EVAR [28].

EVG wall deformations are insignificant due to the high material stiffness; indeed, our results show that the
maximum graft deformation is less than 1 mm. However, EVG wall deformation is a main factor when determining the sac pressure [26,32]. Furthermore, even relatively small, repetitive EVG-wall deformations may result in interactions between the metallic wire and the interwoven graft material, leading to fabric abrasion and holes [30]. In addition, transient solid-fluid interactions can lead to material fatigue and hence device failure. Clinically, that includes metallic fracture (metal wire fracture, stent-strut, and barb/brook breakage), graft fabric holes and suture breakage, and/or separation. Additionally, device corrosion has also been observed clinically. So far, there is no model which can predict such device deteriorations [33]. The reason is the serious lack of time-dependent clinical data due to short follow-ups, especially for the second generation of stent-grafts. Another reason is that most of the patients have been asymptomatic and have not as of yet needed interventions for device fatigue [34]. In this study, we did not consider device failure cases. However, it is possible to simulate device fatigue employing a fluid-structure interaction solver for a realistic stented AAA model in order to assess such problems.

Endoleaks were not considered in this paper. However, in cases of a loose neck attachment, graft defect and/or minor branch backflows, blood may leak into the AAA sac after EVAR. For example, Zarins et al. [37] indicated that endoleaks were diagnosed with CT scans in 38% of 398 patients. Endoleaks may cause an increase in sac pressure and hence higher stresses in the AAA wall. Serious endoleaks can result in EVR failure, AAA rupture, and the need for second procedures. In a preliminary study, we found that if an endoleak volume of 3% is added to the sac volume, the sac pressure and AAA wall stress can increase by 60% [32].

Even though EVG placement may reduce significantly the sac pressure and wall stress, the hemodynamics incurs a drag force which may trigger EVG migration. It is shown that the risk of migration can be high for patients with severe hypertension (see Fig. 9). EVG migration is a common problem for EVAR patients. For example, Zarins et al. [29] reported that the migration rate was up to 8.4% for their 1119 patients within 12 months after EVAR. Serious EVG migration can cause endoleaks, EVG twisting or kinking and hence EVG failure. In this paper, the maximum EVG drag force is about 2 N because the selected EVG size is relative small (17 mm). However, our preliminary research results indicated that the EVG drag force can exceed 5 N for AAAs with a large neck angle, iliac angle, large EVG size, and aorto-unii-iliac EVG [32]. Thus, the fixation of self-expandable or balloon-expandable EVG contact may be inadequate to withstand the pulling forces caused by net momentum changes inside the endovascular graft. Means of extra fixation should be taken into account whenever possible. Concerning the supportive literature, Lifman et al. [12] and Morris et al. [13] confirmed that the drag force increases with EVG size. Mohan et al. reported that the drag force is non-linearly increasing with iliac angle [14]. Sternbergh et al. [35] declared that the migration rate increases by 30% for AAAs with neck angles greater than 40°.

Interestingly, compared with open surgery, the mortality of EVAR is almost the same. However, the key advantages of EVAR are reduced morbidity, shorter hospitalisation, and quicker recovery; but, these benefits may be offset by the cost of the device, the need for continuous control measurements, and eventually the need for late intervention or conversion to open repair [31].

It should be noted that in this study the AAA wall was assumed to be smooth. Intraluminal thrombus and small branches were not considered. In patient-specific stented AAAs, intraluminal thrombics and small branches may affect the fluid-structure interactions, sac pressure and hence possible EVG migration. In the range of pressure loads from 80 to 120 mmHg, linear elastic property values are generally used [3,6,7]. However, non-linear wall properties may provide even more realistic results [5,9,36]. Considering the difficulty to model an actual EVG, i.e., a Nitinol-wire mesh interwoven with synthetic graft material, presently the EVG is assumed to be a uniform shell made of an equivalent composite material. Furthermore, the blood particle effects on the wall were not considered. In a future study, patient-specific CT scan models, non-linear material properties, impact of an intraluminal thrombus, blood particle effects, and additional EVG configurations will be considered.

In summary, this study provides a new technique, i.e., fully coupled fluid-structure interaction, to evaluate the impact of EVG placement, determine wall stresses and sac pressure values, calculate EVG migration forces, and provide physical insight into the biomechanics of stented AAAs. To the authors’ knowledge, this is the first computational FSI study of a stented AAA. Additional clinical applications, including optimal EVG designs, minimizing sac pressure magnitude, preventing endoleaks, and EVG migration, will be performed as future work.

References


