Computational modelling of endoleak after endovascular repair of abdominal aortic aneurysms

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SUMMARY
To better understand the effects of endoleak on the aneurysm intrasac pressure, previously, in a small number of studies, numerical models have been developed for the simulation of endoleak based on coupled fluid–structure interaction (FSI). Although such models can provide useful information on the fluid dynamics and structural deformations in aneurysms after endovascular repair (EVAR), they may computationally be too expensive if one is solely interested in the effects of endoleak on the risk of aneurysm rupture. To assess the value of computationally more efficient lumped parameter models in the simulation of endoleak, in the current study, a lumped parameter-based computational model of an incompletely excluded abdominal aortic aneurysm is developed as well as one based on coupled FSI. Both models are used to study the aneurysm intrasac pressure as a function of the degree of endoleak and the stent-graft compliance. Based on the agreement of the results of the two models it is concluded that the lumped parameter modelling method provides a useful alternative to coupled FSI for modelling the aneurysm intrasac pressure in the presence of endoleak. The modelled pressure could be used as a boundary condition for solid stress analyses to predict the risk of aneurysm rupture after EVAR. Copyright © 2009 John Wiley & Sons, Ltd.

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1. INTRODUCTION

The outcome of endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms (AAAs) is compromised by the possible occurrence of endoleak [1]. In EVAR, to reduce the risk of aneurysm rupture, the aneurysmal part of the vessel wall is excluded from the circulation by means of a stent-graft. Endoleak is generally defined as persistent flow between the stent-graft and the excluded vessel wall, and can be classified according to the origin of the flow [2, 3]. Since all types of endoleak lead to continued pressurization of the aneurysmal sac [4], endoleak is assumed responsible for continued risk of rupture. For endoleak related to the stent-graft attachment sites (type I) and endoleak related to stent-graft failure and disconnection (type III), this effect has been demonstrated in a clinical study [5]. On a patient-specific basis, however, the nature and significance of endoleak remain poorly understood [1].

To better understand the effects of endoleak on the aneurysm intrasac pressure, in a small number of studies, numerical models have been developed for the simulation of endoleak based...
on coupled fluid–structure interaction (FSI). In a study by Li and Kleinstreuer [6], endoleak was represented by small changes of the fluid volume contained by the aneurysmal sac. In this study, it was concluded that for small endoleaks, the intrasac pressure is greatly increased compared with full exclusion. In another study by Li and Kleinstreuer [7], endoleak was represented by flow through a physical connection of the aneurysmal sac with the aortic lumen. In this study, it was concluded that relatively large endoleaks yield intrasac pressures close to the systemic pressure. Based on various simulations, the above authors further concluded that in the absence of endoleak, intrasac pressure pulsatility can still be present due to interactions between the luminal blood flow, the stent-graft, the blood comprised by the aneurysm, and the aneurysm wall [6, 8].

Although FSI-based models can provide useful information on the fluid dynamics and structural deformations in aneurysms after EVAR, such models may computationally be very expensive. In the simulation of endoleak using a weakly coupled method as commonly applied in the simulation of cardiovascular problems (e.g. [9]), numerical instabilities as those observed in the modelling of flow through long compliant vessels [10] are to be expected. As a result, numerical relaxation of the FSI algorithm is required, that especially for small endoleaks can drastically increase the CPU time needed (see also [7]). The effects of endoleak on the risk of aneurysm rupture, however, could possibly also be determined using only a solid stress model of the aneurysm wall. For AAAs before treatment, the peak wall stress computed using such models and the occurrence of rupture were shown to be highly related (e.g. [11–13]). For excluded AAAs in the presence of endoleak, the pressure load applied in such models should be based on the intrasac pressure. This pressure could be determined using a lumped parameter model describing the relation between the systemic and intrasac pressures as a function of the degree of endoleak, the stent-graft compliance, and the compliance of the aneurysm wall [14]. To this end, however, it should first be determined to what extent lumped parameter models are able to capture the effects of three-dimensional FSI on the aneurysm intrasac pressure in the presence of endoleak as observed in more elaborate models.

To assess the value of lumped parameter models in the simulation of endoleak, in the current study, a lumped parameter-based computational model of an incompletely excluded AAA is developed as well as one based on coupled FSI. The FSI model consists of equations describing the structural deformation of the vessel wall and stent-graft and the blood flow in the endoleak lumen and aneurysmal sac, combined with a solution procedure dedicated to deal with numerical instabilities associated with the simulation of small endoleaks. The lumped parameter model is based on a representation of endoleak using viscosity dominated flow in which fluid inertia is neglected to simulate typical endoleak flow rates. Both models are used to study the aneurysm intrasac pressure for different degrees of endoleak and different values of the stent-graft compliance. Based on the agreement of the results of the two models, it is determined whether the lumped parameter modelling method provides a useful alternative to coupled FSI for modelling the aneurysm intrasac pressure in the presence of endoleak and ultimately predicting the risk of aneurysm rupture after EVAR.

2. METHODS

2.1. Geometrical model

The incompletely excluded AAA is modelled as a sinusoidally shaped dilatation of a straight vessel with an inserted cylindrical stent-graft (see Figure 1). The problem is considered symmetrical with respect to both the mid-axial plane and the vessel central axis. At the attachment site, the stent-graft and the aortic wall are separated by an annular region representing the lumen of an endoleak. The vessel dimensions are chosen in the anatomic range, with inflow and maximal diameters of 20 mm and 50 mm, respectively, a total aneurysm length of 80 mm, and a vessel wall thickness of 2.0 mm. The stent-graft wall thickness and the endoleak lumen width each are defined as 0.5 mm. The endoleak lumen length is 10 mm.
Figure 1. Geometry of the incompletely excluded AAA model. The structural domain of the model comprises the stent-graft domain \( \Omega_g \), enclosed by the boundaries \( \Gamma_{g,i}, \Gamma_{g,o}, \Gamma_{g,p}, \) and \( \Gamma_{g,m} \), and the vessel wall domain \( \Omega_w \), enclosed by \( \Gamma_{w,i}, \Gamma_{w,o}, \Gamma_{w,p}, \) and \( \Gamma_{w,m} \). The fluid domain comprises the endoleak domain \( \Omega_l \) and the aneurysmal sac domain \( \Omega_s \). The fluid domain as a whole is enclosed by \( \Gamma_{l,i}, \Gamma_{l,o}, \Gamma_{l,p}, \) and \( \Gamma_{l,m} \). (Subscripts: \( g \) - stent-graft, \( w \) - wall, \( l \) - endoleak, \( s \) - sac, \( i \) - inner, \( o \) - outer, \( p \) - proximal, \( m \) - mid-axial; \( r \) and \( z \) denote the radial and axial coordinates.)

2.2. Fluid–structure interaction model

In the FSI model, the deformation of the structural part of the model comprising the stent-graft and the vessel wall is described using the equations of motion and continuity. In the absence of body forces, for an incompressible material, these equations read

\[
\nabla \cdot \sigma = 0 \\
\det(F) - 1 = 0 \quad \text{in } \Omega_g \cup \Omega_w
\]

with \( \sigma \) the Cauchy stress tensor, and \( F = (\nabla \otimes \mathbf{x})^T \) the deformation gradient tensor with respect to the reference state, where \( \mathbf{x} \) is the spatial coordinate. The constitutive behaviour of the stent-graft and the vessel wall is described using a linearly elastic neo-Hookean material model:

\[
\sigma = -pI + G(B - I), \quad B = FF^T
\]

with \( p \) the hydrostatic pressure, \( I \) the unit tensor, \( B \) the Finger tensor, and \( G \) the shear modulus. The stent-graft and vessel wall mid-axial cross-sections and the proximal wall end are constrained in the axial direction. At the endoleak lumen, the local stent-graft boundary is rigidly coupled to the local wall boundary. At the stent-graft inner boundary, the normal stress is prescribed in terms of a given systemic pressure \( p_{sys} \). At the fluid/solid boundaries, the normal stress is prescribed in terms of the local fluid pressure \( p_f \):

\[
x_z = x_{z,0} \quad \text{on } \Gamma_{g,m} \cup \Gamma_{w,m} \cup \Gamma_{w,p} \\
\nabla_0 \mathbf{x} = \nabla_0 \mathbf{x}_{\Gamma_{l,w}} \quad \text{on } \Gamma_{l,g} \\
\sigma \cdot \mathbf{n} = -p_{sys} \mathbf{n} \quad \text{on } \Gamma_{g,i} \\
\sigma \cdot \mathbf{n} = -p_f \mathbf{n} \quad \text{on } \Gamma_{g,o} \cup \Gamma_{w,i}
\]

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with \( \mathbf{n} \) the boundary outward normal, \( x_z \) the axial coordinate, and \( x_{z,0} \) the axial coordinate in the undeformed situation. If a variable is not prescribed on a certain boundary, a homogeneous Neumann condition is assumed.

The flow in the endoleak lumen and the aneurysmal sac is described using the arbitrary Lagrangian–Eulerian (ALE) formulation of the Navier–Stokes equations. In the absence of body forces, for an incompressible fluid, these equations read

\[
\frac{\partial \mathbf{v}}{\partial t} + \rho(\mathbf{v} - \mathbf{w}) \cdot \nabla \mathbf{v} - \nabla \cdot \mathbf{\sigma} = 0 \quad \text{in } \Omega_t \cup \Omega_s
\]

\[
\nabla \cdot \mathbf{v} = 0
\]

where \( t \) denotes the time, and with \( \mathbf{v} \) the fluid velocity, \( \mathbf{w} \) the velocity of the fluid domain, and \( \rho \) the density. The constitutive behaviour of the blood is described using an inelastic Newtonian model:

\[
\mathbf{\sigma} = -p \mathbf{I} + 2\eta \mathbf{D}, \quad \mathbf{D} = \frac{1}{2} [\nabla \mathbf{v} + (\nabla \mathbf{v})^T]
\]

with \( p \) the fluid pressure, \( \mathbf{D} \) the rate of deformation tensor, and \( \eta \) the viscosity. At the endoleak lumen proximal end again the normal stress is prescribed in terms of the given systemic pressure \( p_{\text{sys}} \), and the radial velocity is constrained to the mesh velocity. At the sac mid-axial cross-section, the fluid velocity is constrained in the axial direction, and at the fluid/solid boundaries, a no-slip condition is prescribed:

\[
(\mathbf{\sigma} \cdot \mathbf{n}) \cdot \mathbf{n} = -p_{\text{sys}} \quad \text{on } \Gamma_{1,p}
\]

\[
v_r - w_r = 0 \quad \text{on } \Gamma_{s,m}
\]

\[
v_z = 0 \quad \text{on } \Gamma_{g,0} \cup \Gamma_{g,i}
\]

\[
\mathbf{v} - \mathbf{w} = 0 \quad \text{on } \Gamma_{g,0} \cup \Gamma_{g,i}
\]

with \( r \) the radial coordinate. Again, if a variable is not prescribed on a certain boundary, a homogeneous Neumann condition is assumed. The fluid domain velocity \( \mathbf{w} \) is derived from the fluid domain displacement, which is computed using a compressible linearly elastic deformation problem as described in Van de Vosse et al. [9], using the wall displacement as boundary condition.

2.3. Fluid–structure coupling

The equations of the FSI model are solved in a weakly coupled approach (see Figure 2). At initialization, the fluid velocity \( \mathbf{v} \) and the mesh velocity \( \mathbf{w} \) are set to zero, and the fluid pressure \( p \) is set to the systemic mean pressure \( \bar{p}_{\text{sys}} \). By pressurization of the structural model, the initial coordinates \( x_0 \) are determined. Next, every timestep, first, the structural problem is solved to give the wall displacement. Next, the fluid mesh displacement is computed by solving the linearly elastic deformation problem defined on the fluid domain. Finally, the fluid problem is solved, providing the fluid velocity and pressure distributions on the deforming mesh.

To account for numerical instabilities associated with the fluid–structure coupling, the different solution steps are combined in an iterative loop for underrelaxation of the mesh velocity. In this loop, at every iteration, the mesh deformation and the associated mesh velocity \( \mathbf{w} \) are determined for a given estimate of \( p \). Next, iteratively, without the adjustment of \( \mathbf{w} \), the new fluid problem solution and the updated mesh coordinates are determined. The loop is set to have converged when the error of \( p \) with respect to the initial estimate \( p^e \) is less than a criterion \( \varepsilon_p \). Next, based on the estimated mesh velocity \( \mathbf{w}^e \) set at the beginning of the timestep, the convergence of \( \mathbf{w} \) is checked against a criterion \( \varepsilon_w \). The iterative loop for the pressure solution is repeated until \( \mathbf{w} \) has converged. Underrelaxation of \( \mathbf{w} \) is applied at every first iteration.

2.4. Lumped parameter model

The lumped parameter model consists of a resistor representing the flow resistance of the endoleak lumen (\( R \)), and two capacitors, respectively, accounting for the compliance of the stent-graft (\( C_g \)).
and the compliance of the vessel wall ($C_w$) (see Figure 3). Based on this model, the intrasac pressure $p_{sac}$ can be defined as a function of the systemic pressure $p_{sys}$. When the signals are expressed as discrete Fourier series:

$$p_{sac} = \sum_{n=0}^{N} \hat{p}_{sac,n} e^{in\omega} \quad p_{sys} = \sum_{n=0}^{N} \hat{p}_{sys,n} e^{in\omega}$$

(7)

with $\hat{p}_{sac,n}$ and $\hat{p}_{sys,n}$ the complex Fourier coefficients of $p_{sac}$ and $p_{sys}$, respectively, $N$ the number of harmonics, $n$ the harmonic number, $\omega$ the fundamental frequency, and $i$ the imaginary number, the relation between $p_{sac}$ and $p_{sys}$ be written as

$$\frac{\hat{p}_{sac,n}}{\hat{p}_{sys,n}} = \frac{1+i\omega RC_g}{1+i\omega (C_g + C_w)}$$

(8)
Figure 3. Lumped parameter model of the incompletely excluded AAA. The parameters $R$, $C_g$, and $C_w$, respectively, denote the endoleak resistance, the stent-graft compliance, and the compliance of the vessel wall. The variables $p_{sys}$ and $p_{sac}$, respectively, denote the systemic pressure and the intrasac pressure.

Figure 4. Prescribed systemic pressure (after [16]).

In this definition it is assumed that the represented endoleak situation is in periodic state, meaning that both $p_{sac}$ and $p_{sys}$ are time-periodic.

The parameter values of the model are based on geometrical and physical assumptions. The stent-graft compliance and the compliance of the vessel wall are based on the variational area-to-pressure relation of thin-walled tubes

$$\frac{\partial A}{\partial p} = \frac{2\pi a^3(1-\nu^2)}{Eh}, \quad E = 2(1+\nu)G$$

with $A$ the tube cross-sectional area, $p$ the internal pressure, $a$ the tube radius, $\nu$ the wall material Poisson ratio, $E$ the elastic modulus, and $h$ the wall thickness. The compliance values are determined by integrating (9) along the central axis as depicted in Figure 1. The resistance of the endoleak lumen is based on the pressure-flow relation for axial flow through an annulus:

$$R = \frac{(P_0 - P_L)}{Q} = \left(\frac{\pi a^4}{8\eta L} \left(1 - \kappa^4 + \frac{(1-\kappa^2)^2}{\ln \kappa}\right)\right)^{-1}, \quad \kappa = \frac{r_i}{r_o}$$

with $(P_0 - P_L)$ the pressure drop, $Q$ the volume rate of flow, $L$ the annulus length, and $r_i$ and $r_o$ the annulus inner and outer radii, respectively [15].

2.5. Implementation and simulations

For the FSI model, the intrasac pressure to be compared with the lumped parameter model response for a given systemic pressure is defined as the pressure computed in a central point inside the aneurysm, located at the centre of the mid-axial boundary $\Gamma_{x,m}$. The equations of the FSI model are linearized, discretized and solved using the SEPRAN finite element package (SEPRA BV, The Hague, NL). The lumped parameter model response is the intrasac pressure as computed using (8), where the Fourier representation of the systemic pressure signal and the different element parameter values defined by (9) and (10) are used as input. The response is computed using Matlab (The MathWorks, Inc., Natick, MA). The systemic pressure is based on the pressure in the healthy aorta according to Olufsen et al. [16] (see Figure 4). The signal is represented using 11 harmonics.
The intrasac pressure is obtained for seven different degrees of endoleak and five different values of the stent-graft compliance. The degree of endoleak is varied by varying the resistance of the endoleak lumen through adjustments to the local viscosity. The stent-graft compliance is varied by adapting the stent-graft shear modulus. For the initial cases, the two parameters equal, respectively, the viscosity of the blood and the shear modulus of the vessel wall. For the other cases, both parameters are multiplied by increasing powers of two. The viscosity and density of the blood are set to, respectively, 3.5 mPa s and 1080 kg/m³ [17]. With these properties, the endoleak viscosity varies between 3.5 mPa s (low resistance) and 220 mPa s (high resistance). The stent-graft and the vessel wall both are considered incompressible, such that \( \nu = 0.5 \). The shear modulus and the elastic modulus of the vessel wall are set to 1.0 and 3.0 MPa, respectively, based on an elastic modulus of around 3 MPa applied by Di Martino et al. [18]. As a result, the stent-graft shear modulus varies between 1.0 and 16 MPa. With these values, the stent-graft compliance as defined by (9) varies between 2.3 mm²/kPa (high compliance) and 0.14 mm²/kPa (low compliance). As an illustration, for the compliance of regular stent-grafts, defined in terms of the change of diameter, values of up to 2.0% per 100 mm Hg are reported [19]. For the current geometry, this corresponds to 0.76 mm²/kPa.

For each of the degrees of endoleak, a number of time periods are simulated until the endoleak situation has stabilized. The situation is considered stable when for a certain simulated period, the intrasac mean pressure as compared with the previous period does not vary by more than 1.0 Pa. To limit the simulation time, the solution of the fluid problem for a certain viscosity case is used as the initial condition for the following case. Further, the relaxation factor applied for the underrelaxation of the mesh velocity is varied. For the first three viscosity cases, a relaxation factor of 0.05 is used. For the other cases, this value is repeatedly halved. The convergence criteria for the pressure and mesh velocity solutions as described in the appendix are 0.5 Pa and \( 0.5 \times 10^{-3} \) mm/s, respectively.

3. RESULTS

For the studied values of the endoleak resistance and the stent-graft compliance, the lumped parameter model yields a good approximation of the intrasac pressure as computed by the FSI model (see Figure 5). For high stent-graft compliance (see Figure 5(a)), the intrasac pressure at low values of the endoleak resistance approximates systemic values. When the resistance is increased, the intrasac pressure is attenuated, while the pressure signal largely remains of the same shape. For higher resistance, there is better agreement between the lumped parameter model results and the results of the FSI model. For low stent-graft compliance (see Figure 5(b)), the intrasac pressure at low endoleak resistance again approximates systemic values. When the resistance is increased, the intrasac pressure signal is both attenuated and subjected to a change of shape. In this case, the attenuation is larger than in the case of high stent-graft compliance. For the FSI model, the intrasac pressure at high endoleak resistance is slightly decreased as compared with the lumped parameter model.

Also the variation of the endoleak flow rate for different values of the endoleak resistance and stent-graft compliance as computed by the FSI model is well approximated by the lumped parameter model (see Figure 6). For both high and low stent-graft compliance, the flow rate is decreased with an increase in the endoleak resistance. For low compliance, however, the flow rate at higher values of the endoleak resistance is increased as compared with the case of high compliance. For both cases, for higher endoleak resistance, there is slightly better agreement between the lumped parameter model results and the results of the FSI model.

The agreement of the lumped parameter model results with the results of the FSI model is reflected by the transfer function of the systemic pressure to the intrasac pressure (see Figures 7 and 8). With an increase in the endoleak resistance, the modulus of the transfer function, describing the amplification of the intrasac pressure signal as compared with the systemic signal, is decreased for all higher harmonics. The minimal level attained by the different moduli is lower for low
Figure 5. Intrasac pressure for different values of the endoleak resistance in the FSI model (dashed) and lumped parameter model (solid) for high and low stent-graft compliance. The dotted line depicts the systemic pressure: (a) high stent-graft compliance and (b) low stent-graft compliance.

There is better agreement between the modulus of the lumped parameter model and that of the FSI model for higher resistance (see Figure 7). Further, with an increase in the endoleak resistance, the phase of the transfer function, describing the time shift of the intrasac pressure signal, is decreased for the lower harmonics. For the higher harmonics, the phase initially is decreased, after which it is increased again. For low stent-graft compliance, the maximal phase shift is larger than for the case of high compliance. Yet, fluctuations occurring at some of the higher harmonics for the FSI model are also larger. In general, there is better agreement between the phase of the lumped parameter model and that of the FSI model for higher endoleak resistance, except for when the resistance is maximal (see Figure 8).

By an analysis of the intrasac pressure in terms of its mean and amplitude it can be seen that the differences occurring between the lumped parameter model results and the results of the FSI model themselves are dependent on the endoleak resistance and stent-graft compliance as well (see Figure 9). For the intrasac pressure mean, irrespectively of the endoleak resistance, the value predicted by the lumped parameter model equals the systemic mean. As predicted by the FSI model, however, for higher values of the endoleak resistance, the intrasac pressure mean is
lower. The deviation from the systemic mean further is dependent on the stent-graft compliance. With respect to the intrasac pressure amplitude, irrespectively of the stent-graft compliance, the agreement between the results of the two models is better for higher resistance.

4. DISCUSSION

The FSI model and the lumped parameter model both predict a decrease in the intrasac pressure amplitude and a decrease in the endoleak flow rate for increasing endoleak resistance. These findings are consistent with the findings of the FSI-based study by Li and Kleinstreuer [7], who report a decrease in the intrasac peak pressure and a decrease in the endoleak flow rate for smaller endoleak apertures. The findings further agree with experimental studies in which endoleak was found to lead to intrasac pressure pulsatility proportionally related to the flow rate [14, 20–22]. According to the lumped parameter model for all cases studied and according to the FSI model for low endoleak resistance, the degree of endoleak does not seem to influence the intrasac mean pressure. As observed previously in both animal studies [20, 23] and studies using experimental
Figure 7. Modulus of the transfer function of the systemic pressure to the intrasac pressure for different values of the endoleak resistance in the FSI model (dashed) and lumped parameter model (solid) for high and low stent-graft compliance: (a) high stent-graft compliance and (b) low stent-graft compliance.

setups [14, 22], for the cases stated, endoleak yields an intrasac mean pressure that approximates the systemic mean pressure irrespective of the associated flow rate.

For an increase in the stent-graft compliance, the computational models predict a decrease in the intrasac pressure amplitude at low endoleak resistance, an increase in the intrasac pressure amplitude at high endoleak resistance, and associated changes of the endoleak flow rate. The increased pressure amplitude agrees with the findings in [6], where in the absence of endoleak, the intrasac pressure versus systemic pressure ratio was found to be decreased for an increasing elastic modulus of the stent-graft. Similar findings were reported in [8]. From the synchronicity of the intrasac pressure signal at high stent-graft compliance and maximal endoleak resistance with the systemic pressure signal, it can be gathered that the increase in the intrasac pressure amplitude is due to transmission of the systemic pressure pulse to the aneurysmal sac by deformation of the stent-graft. At low stent-graft compliance, the intrasac pressure signal is mainly dependent on transmission through the endoleak lumen. From the intrasac pressure signal for this case, it can be deduced that this type of transmission is concerned with significant changes to the signal shape.

From the improved agreement of the lumped parameter model results with the results of the FSI model for higher endoleak resistance, it can be gathered that the lumped parameter model
Figure 8. Phase of the transfer function of the systemic pressure to the intrasac pressure for different values of the endoleak resistance in the FSI model (dashed) and lumped parameter model (solid) for high and low stent-graft compliance: (a) high stent-graft compliance and (b) low stent-graft compliance.

in the current configuration is most suited for the simulation of small endoleaks. For the larger endoleaks simulated, where the Reynolds number can obtain values of up to 50, the assumption of viscosity-dominated flow will not always be valid. Therefore, the deviations between the results of the two models at low endoleak resistance presumably are due to neglect of the inertance of the fluid inside the endoleak lumen and aneurysmal sac. This neglect becomes apparent by the relative overshoot of the endoleak flow rate in the FSI model, and relatively low values of the pressure transfer function modulus for the high frequencies in the lumped parameter model. The deviations further can be due to neglect of the flow resistance of the fluid inside the aneurysmal sac. The latter mostly becomes clear from a relative shift of the transfer function phase. Deviations of both kinds decrease for increasing endoleak resistance due to increasingly dominant viscous flow through the endoleak lumen. To optimize the agreement for large endoleaks, the lumped parameter model should be extended with an inductor in parallel to the main resistor to represent the inertance of the endoleak, and an additional resistor and inductor to represent the flow resistance and inertance of the fluid inside the aneurysmal sac.

The deviations of the intrasac mean pressure in the FSI model as compared with that in the lumped parameter model and the deviations of the pressure transfer function phase for high values of the endoleak resistance denote that for the simulation of small endoleaks the lumped parameter
model is better suited than the FSI model. The deviations are likely due to the fact that the applied convergence criteria used to check for a time periodic solution are too large with respect to the applied relaxation factors. A more accurate solution could be obtained using more stringent criteria, at the cost of computational efficiency. Owing to inherent instability of the weak fluid–structure coupling of the FSI problem solution procedure, underrelaxation of the mesh velocity, however, remains necessary. The instability is due to the fact that in the current implementation, during the iterative procedure to obtain complementary solutions for the structural problem and the fluid problem, the intermediate solutions of the latter need to comply to the conservation of mass. As a result, at low endoleak resistance, relatively small variations of the wall displacement can lead to significant variations of the endoleak flow. At high resistance, the endoleak flow, however, is limited. The pressure variations resulting from this limitation give rise to the numerical instabilities also observed in the modelling of flow through long compliant vessels [10]. The problem is more serious for higher endoleak resistance, and cannot be solved by selecting a smaller timestep. More stable computations at lower computational cost could possibly be achieved by the implementation of a solution procedure that is fully coupled.
Based on the agreement of the results of the two models, it is concluded that the lumped parameter modelling method provides a useful alternative to the coupled FSI for modelling the aneurysm intrasac pressure in the presence of endoleak. The modelled pressure could be used as boundary condition for solid stress analyses to predict the risk of aneurysm rupture after EVAR. To obtain similar stresses in the solid stress analysis as in the FSI analysis, the solid stress model should include the stent-graft, and be subject to the same kinematic boundary conditions as the FSI model. The absence of pressure gradients as those implicitly present in the FSI analysis is not expected to influence the results. The maximal pressure gradient is in the order of $\rho v^2$, with $v$ the maximal endoleak flow velocity. For the highest degree of endoleak, this property amounts to around 100 Pa, or less than 1% of the peak systolic pressure. It should be noted, however, that the models developed in this study are intended to give a phenomenological representation of endoleak rather than one that is anatomically and physiologically realistic. In reality, type I endoleak is not likely to occur along the full circumference of the inserted stent-graft. Further, the intrasac pressure will also depend on factors not included in the models, such as endoleak thrombosis and the presence of thrombus inside the aneurysmal sac, the presence of an outflow tract connected to the sac, and the occurrence of other types of endoleak. With respect to the vessel wall and the stent-graft, the current models do not incorporate realistic geometries, non-homogeneous mechanical properties, or initial stress. The extent to which including these details could help to refine the FSI and lumped parameter models should be subject to further study. Also, in future studies, attention should be paid to the validation of the predictive value of the computed stresses for rupture of AAAs in the presence of endoleak. The relation between peak stress and rupture as obtained previously for AAAs before treatment may be affected by including the stent-graft in the solid stress analysis. Currently, however, due to the comparative nature of the study, the above shortcomings are of limited importance.

5. CONCLUSION

In the current study, a lumped parameter-based computational model of an incompletely excluded AAA has been developed as well as the one based on coupled FSI. Both models predict a decrease in the intrasac pressure amplitude and a decrease in the endoleak flow rate for increasing endoleak resistance. For an increase in the stent-graft compliance, the models predict a decrease in the intrasac pressure amplitude at low endoleak resistance, an increase in the intrasac pressure amplitude at high endoleak resistance, and associated changes of the endoleak flow rate. Based on the agreement of the results of the two models, it is concluded that the lumped parameter modelling method provides a useful alternative to coupled FSI for modelling the aneurysm intrasac pressure in the presence of endoleak. The modelled pressure could be used as boundary condition for solid stress analyses to predict the risk of aneurysm rupture after EVAR.

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