Analysis of biomechanical factors affecting stent-graft migration in an abdominal aortic aneurysm model

Z. Li\textsuperscript{a}, C. Kleinstreuer\textsuperscript{b,*}

\textsuperscript{a}Department of Mechanical and Aerospace Engineering, North Carolina State University, Raleigh, USA
\textsuperscript{b}Department of Mechanical and Aerospace Engineering and Department of Biomedical Engineering, North Carolina State University, Raleigh, USA

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Abstract

Focusing on a representative abdominal aortic aneurysm (AAA) with a bifurcating stent-graft (SG), a fluid–structure interaction (FSI) solver with user-supplied programs has been employed to solve for blood flow, AAA/SG deformation, sac pressure and wall stresses, as well as the downward forces acting on the SG. Simulation results indicate that implanting a SG can significantly reduce sac pressure, mechanical stress, pulsatile wall motion, and maximum diameter change in AAAs; hence, it may restore normal blood flow and prevent AAA rupture effectively. The transient SG drag force is similar in trend as the cardiac pressure. Its magnitude depends on multi-factors including blood flow conditions, as well as SG and aneurysm geometries. Specifically, AAA neck angle, iliac bifurcation angle, neck aorta-to-iliac diameter ratio, SG size, and blood waveform play important roles in generating a fluid flow force potentially leading to SG migration. It was found that the drag force can exceed 5 N for an AAA with a large neck or iliac angle, wide aortic neck and narrow iliac arteries, large SG size, and/or abnormal blood waveform. Thus, the fixation of self-expandable or balloon-expandable SG contact may be inadequate to withstand the forces of blood flowing through the implant and hence means of extra fixation should be considered. A comprehensive FSI analysis of the coupled SG–AAA dynamics provides physical insight for evaluating the luminal hemodynamics, and maximum AAA-stresses as well as biomechanical factors leading potentially to SG migration.

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1. Introduction

An aneurysm is an irreversible dilation, i.e., ballooning, of an artery greater than 50% of its nominal diameter, due to gradual wall weakening. As the aneurysm expands, it may eventually rupture. Traditionally, the available treatment required open surgical repair. Specifically, open surgery involves a large incision and replacement of the diseased aorta segment with a synthetic prosthetic graft made of polyester or ePTFE. Alternatively, endovascular aneurysm repair (EVAR) is a minimally invasive technique to treat aneurysms. In EVAR, a stent-graft (SG) is guided from the femoral artery to the affected artery segment in order to shield the aneurysm from the blood pressure, eliminate blood circulation in the aneurysm intra-sac, and hence prevent wall rupture. Thus, the primary objective is to exclude the aneurysm cavity (or intra-sac) completely from the impact of the pulsatile blood flow. However, EVAR is a relatively new technology without long-term follow-up outcome. While it has shown outstanding success for patients with abdominal aortic aneurysms (AAAs), it can also cause many problems, such as seepage of blood into the cavity (endoleaks), SG migration, SG failure, and other complications. These
problems, especially the specter of SG migration, are all closely related to hemodynamics and biomechanics. Migration usually means distal movement of the proximal portion of the stent (see Fig. 1). It may be thought of as a failure of attachment with an unchanged aorta or as a failure related to changes in aneurysm morphology. If the neck is too short, the attachment is marginal or if there is insufficient columnar strength, migration may occur. Zarins et al. (2003) reported that the SG migration rate was about 18.8% in 1119 patients after 3 years follow-up of EVAR. Based on statistics for clinical data from the UNC Vascular Registry and Washington University Vascular Surgery Database in St. Louis, we found that the migration rate during 40 months after EVAR was about 16.7% (Li, 2005). Migration is associated with neck dilatation in 50% of all cases (Sternbergh et al., 2004). Factors that help to maintain SG position include: friction, barb or hook penetration, suprarenal attachment, column strength, and arterial ingrowth. Malina et al. (1998) discussed the effects of hooks and barbs and indicated that they may increase the fixation of SGs; but, they also can cause complications during SG placement and in case of diseased aortic necks. Typically, fixation forces for SGs without hooks or barbs are 5–6 N (Lawrence-Brown et al., 1999). Resch et al. (2000) performed experiments with a cadaveric aorta to study the impact of SG designs on proximal SG fixation in AAAs, where the median fixation forces were 4.5 N for Talent, 9.0 N for Vanguard, 12.5 N for Ancure (barbed), 24 N for Zenith (barbed) and 25 N for Palmaz (barbed). Lambert et al. (1998) assessed experimentally proximal SG fixation in infra-renal aortas. They found that there is a theoretical advantage of oversizing an aortic prosthesis and that sufficient anchorage is achieved in an aortic neck of 10 mm to prevent migration when fully deployed. Albertini et al. (2001) concluded that neck angulation was the risk factor most significantly related to endoleaks and graft migration. In an in vitro study, Volodos et al. (2003) analyzed factors affecting the displacement force exerted on an SG after AAA repair and declared that systemic hypertension is obviously important and its control may be necessary to prevent migration.

With a very few exceptions (e.g., Scotti et al., 2004; Di Martino et al., 2004; Chong and How, 2004; Gawenda et al., 2004), researchers investigated the dynamics of AAAs and SGs separately (see Vorp et al., 1998; Di Martino et al., 2001; Liffman et al., 2001; Finol et al., 2003; Fillinger et al., 2003; Morris et al., 2004; among others). For example, Gawenda et al. (2004) measured the influence of aneurysm and SG wall compliance on intra sac pressure for in vitro stented aneurysm models, while Chong and How (2004) visualized flow patterns in
a commercial SG. Morris et al. (2004) developed a mathematical model to predict the transient drag force acting on a bifurcated rigid SG without considering the sac blood, AAA wall and SG–AAA interaction dynamics. Finol et al. (2003) analyzed numerically the fluid–structure interaction (FSI) of a nonstented aneurysm model. Scotti et al. (2004) and Di Martino et al. (2004) studied the stresses on the aneurysm sac before and after EVAR as well as the stresses placed on the SG and its attachment system. They reported that the majority of stress is absorbed by the SG after repair reducing AAA wall stresses by 90%.

In this paper, a coupled transient 3-D FSI analysis of a representative stented AAA model is described and key biomechanical factors based on SG–AAA interaction dynamics, potentially leading to SG migration, are discussed.

2. Materials and methods

For the numerical simulation of interacting pulsatile blood flow, bifurcated SG distensibility, stagnant blood, and aneurysm wall motion and stress, two coupled sets of partial differential equations plus associated boundary conditions have to be solved. The governing equations for fluid and structure dynamics are discussed in our previous publications (see Li and Kleinstreuer, 2005a,b). The Quemada blood rheology, as modified by Buchanan et al. (2000), was employed:

$$\eta = \frac{\eta_p}{\left[1 - \frac{1}{4} \left(\frac{k_0 k_\infty}{\kappa H_\text{T}}\right)^{\frac{1}{2}}\right] H_\text{T}^2}. \quad (1)$$

Here, $\eta_p$ is the plasma viscosity, $\gamma_\infty$ is defined by a “phenomenological kinetic model”. $k_0$ is the lower limit Quemada viscosity constant, $k_\infty$ is the upper limit Quemada viscosity constant, and $H_\text{T}$ is the hematocrit. In this simulation, $\rho = 1.050 \text{ g/cm}^3$, $k_0 = 4.58619$, $k_\infty = 1.29173$, $H_\text{T} = 40\%$, and $\eta_p = 0.014 \text{ dyn s/cm}^2$ (Buchanan et al., 2000).

The stented aneurysm model consists of a lumen with pulsatile blood flow formed by a bifurcated elastic SG, stagnant blood and the 3-D elastic aneurysm wall (Fig. 1). Based on CT-scan images from the UNC School of Medicine, we selected a representative CT-scan model. Due to the difficulties determining the AAA/SG wall thicknesses precisely, we employed Amira (San Diego, CA) to convert the surface profile of the CT-scan image to a CAD model. Following the CAD profiles, we used SOLIDWORKS to build the SG, AAA sac and AAA wall, and then imported them into ANSTS in IGES format for the ANSYS solvers. The interacting materials included the luminal blood, SG wall, stagnant blood in the AAA cavity, and the AAA wall. The SG was assumed to be a uniform 3-D bifurcating shell attached to the proximal neck and iliac artery wall. The cavity between SG and aneurysm wall was filled with stagnant blood, experiencing a time-dependent pressure as a result of the dynamic FSIs (Li and Kleinstreuer, 2005a).

To simplify the simulations, the blood flow was assumed to be incompressible and laminar, i.e., blood particle effects were not considered. The cavity was assumed to be filled with stagnant blood, i.e., no endoleaks in the stented aneurysm models. The material properties in the multi-structure sections were assumed to be isotropic and linear. No residual stresses and tissue growth on the walls were considered. The SG neck had a tight contact and no slippage occurred.

It should be noted that several SG geometries were considered, where Table 1 lists the structure parameter values for one case used in the present simulations. When the key parameters of the SG geometry were altered (e.g., the neck angle or main-body diameter), the other parameters were kept unchanged. As installed SG changes, the AAA neck, AAA iliac bifurcation and AAA iliac diameters may also change accordingly. This procedure can be performed in SOLIDWORKS with part connection features. 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 nonaneurysmal artery (Thubrikar et al., 2001). In this paper, the Young’s modulus was assumed to be 4.66 MPa. The healthy artery section (neck) was incompressible with a Poisson ratio of 0.49, and the aneurysm wall was nearly incompressible with a Poisson ratio of 0.45 (Di Martino et al., 2001). For a bifurcated NiTi-stent interwoven with graft material (SG), no direct experimental data was available; thus, an equivalent Young’s modulus for the uniform SG configuration was assumed to be 10 MPa (Suzuki et al., 2001).

The physiologically representative inflow velocity waveform is shown in Fig. 2a 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. 2b), the peak and average pressures are 122 and 98.7 mmHg, respectively (Meter, 2000). The pulse period was chosen as $T = 1.2 \text{s}$. Due to heart or blood vessel diseases, some AAA patients have abnormal blood flow waveforms. Thus, we employed three different iliac exit pressure waveforms to evaluate their impact on the drag force. The inlet velocity profile was uniform and fully developing in an extended tube of five times the neck diameter. For the composite structures, the boundary conditions were a fixed degrees-of-freedom (DOF) at the inlet and the exit, and a free DOF on the wall.

The finite element software package ANSYS Multiphysics for linear and nonlinear analyses in Arbitrary Lagrangian–Eulerian (ALE) formulation has been employed to solve this FSI problem. Specifically, it uses
separately ANSYS FLOTRAN 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. The detailed FSI algorithm was discussed by Li and Kleinstreuer (2005a, b). The sac stagnant blood is special kind of fluid for which ANSYS provides a special fluid element (FLUID80) which is used to model stagnant fluids in containers having no net flow rate. The fluid element is particularly well suited for calculating hydrostatic pressures and fluid/solid interactions. As required by element features, the “fluid elastic modulus” (bulk modulus) is approximately 2000 MPa and the viscosity property is 0.014 dyn s/cm². To simplify the oversizing neck contact, we applied a pre-pressure (about 0.16 MPa for a neck of \( d = 24 \) mm) in the SG neck which can produce 15% of oversize. The pre-stress was used to maintain a tight contact in the neck under different load conditions. The ANSYS surface-to-surface contact element was employed, not considering any device slippage.

It is noted that even though linearly elastic material properties were assumed, the complex and multi-structural geometry of a stented AAA may exhibit nonlinear geometric behavior. Hence, the geometric nonlinear algorithm was used to obtain more accurate results (Wang et al., 2002), considering the geometry nonlinear, large deformation, and dynamic analysis schemes. In geometrically nonlinear analyses, large geometric changes affect the generation of the stress stiffness matrix. Considering that AAA-tissue stiffness increases with strain, the “stress stiffness effect” mode was adopted for the structure solver. This option is provided by the ANSYS nonlinear solver. The drag force exerted on the SG, due to fluid friction and net momentum change, was computed from surface integration of pressure and wall shear stress under physiologically realistic conditions to assess incipient SG migration.

In order to satisfy the criteria for mesh convergence, meshes for both fluid and solid domains were refined until mesh-density independence of the results was achieved. A total of 76,730 8-node fluid elements were needed for meshing the luminal blood flow region, 66,820 structure elements for the SG and AAA wall, and 19,250 elements with bulk modulus of fluid for the stagnant cavity. The number of elements on the FSI interface was 7168. In order to test the accuracy of the coupled fluid–structure solver, several computer model validation studies were performed (see Li and Kleinstreuer, 2005a, b). In this simulation, a variable time step was employed, where \( \Delta t_{\text{min}} = 0.005 \) 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 for transient flow on an IBM p690 workstation.

<table>
<thead>
<tr>
<th>Parameters</th>
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<th>Aneurysm</th>
<th>SG</th>
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<td>1.5–1.0 (Thubrikar et al., 2001)</td>
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<td>Diameter (mm)</td>
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<td>60</td>
<td>Main body diameter: 17 Iliac leg: 11</td>
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<td>Main body: 60</td>
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<td></td>
<td>Iliac artery: 30</td>
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<td>Iliac leg: 70</td>
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<td>Young's modulus (MPa)</td>
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<tr>
<td>Density (g/cm³)</td>
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<td>1.12</td>
<td>Equivalent: 6.0</td>
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</table>

Table 1
Parameters required in the simulation

Fig. 2. Physiological inlet velocity and outlet pressure waveforms for a representative SG.
3. Results

3.1. SG benefits

Obviously, a securely placed SG forms a new blood conduit and shields completely the weakened aneurysm wall from the pulsatile blood flow. Selecting a time level during the critical decelerating phase (see Fig. 3, \( t/T = 0.27 \)), without the SG, two vortices appear near the proximal neck, the average blood pressure in the aneurysm reaches 120.8 mmHg, and the maximum wall stress at the inner wall near the distal end is up to 0.59 MPa, which is very close to the yield stress of 0.67 MPa (Raghavan et al., 1996). The diameter change reaches 2.0 mm at the maximum diameter level. However, after SG placement, blood flows through the new conduit in similar patterns as in a healthy aorta. Still, there are some small vortices in the SG lumen caused by the proximal neck angle and the iliac bifurcation. Now, the sac blood pressure is only 14.38 mmHg, i.e., 11.8% of the current luminal pressure as confirmed by Sonesson et al. (2003). The maximum AAA diameter

\[ p_{\text{max}} = 14.38 \text{ mmHg}, 11.8\% \text{ of } p_{\text{luminal}} \]

Fig. 3. Influence of SG placement on blood flow and AAA wall at peak pressure level \((t/T = 0.27)\).
change is only 0.17 mm. It should be noted, however, that the net force exerted on the SG is 1.97 N. The maximum wall stress decreases from 0.59 to 0.03 MPa; whereas the maximum SG wall stress reaches 1.76 MPa near the junction. Clearly, a proper SG placement can significantly reduce sac pressure and AAA wall stress. Indeed, based on computational finite element models, Scotti et al. (2004) and Di Martino et al. (2004) studied the stresses on the aneurysm sac before and after EVAR as well as the stresses placed on the SG and its attachment system. They reported that the majority of stress is absorbed by the SG after repair reducing AAA wall stresses by 90%.

3.2. Drag force on SG

The drag force acting on the SG includes a downward component, \( F_1 \), and transverse (or sideway) component, \( F_2 \), as shown in Fig. 4, which depicts an SG before insertion. In general, the downward force affects the proximal neck fixation and the sideway force influences the iliac leg fixation; clearly, \( F_1 > F_2 \) and hence we consider the downward force \( F_1 \) as the potential downward drag force \( F \), i.e., \( F \approx F_1 \), potentially causing SG migration. The drag force is generated by the net blood pressure and friction on the SG wall, where pressure is exerted from both sides, i.e., lumen and cavity. However, the cavity pressure is unknown and determined by the FSIs between lumen blood, SG wall, sac blood and AAA wall. Actually, pressure is the major contributor to the drag force because friction is very small. It was found that the wall shear stress contribution is less than 3% (Liffman et al., 2001). In general, the time-dependent SG drag force is, in trend, somewhat related to the cardiac blood pressure (see Fig. 5 vs. Fig. 2b). During one cycle, it varies from 1.3 to 2.1 N in this case study.

As mentioned, there are several biomechanical factors which influence the SG drag force to various degrees. For example, the impact of SG size is very significant. As indicated in Fig. 6, the maximum drag force is only about 2 N when the SG diameter is 18 mm; however, at 32 mm, the force increases to 7 N, exhibiting an almost linear dependence as confirmed by Liffman et al. (2001).

Another influential geometric factor is the SG-body-to-iliac-leg diameter ratio. A nonlinear increase \( \Delta F = 5.3 \) N can be observed when \( d_1 / d_2 \) doubles from 1.5 to 3.0 (Fig. 7). The reason is that larger \( d_1 / d_2 \)-ratios force more blood to converge suddenly into the small daughter vessels, which results in a significant net momentum change. Thus, to decrease the risk of SG migration, the main-body/iliac-leg diameter ratio should be as small as possible.

The iliac angle also influences possible SG migration. As indicated in Fig. 8, when \( \beta = 90^\circ \), \( F = 5.8 \) N, while for \( \beta = 23^\circ \), \( F = 3.8 \) N. The reason is that a large AAA iliac angle results in a large net momentum change (pressure, inertia, and viscous terms). Actually, the increase in the projected surface area of the iliac artery wall onto the transverse plane is the major reason. Our
simulation results are in agreement with the findings of Mohan et al. (2002) and Morris et al. (2004), i.e., that the drag force is nonlinearly increasing with iliac angle. Similarly, the effect of the neck angle \( \alpha \) is negligible when both are less than 40°; however, \( F \) can increase significantly for \( \alpha > 40° \) (Fig. 8). Thus, for a large neck angle, a higher neck fixation force should be considered. Based on clinical observations, Sternbergh et al. (2002) declared that the migration rate increases by 30% for AAAs with neck angles greater than 40°. Clearly, the pressure waveform can change the drag force measurably (Fig. 9). For waveform-I with a sharp systolic slope, \( F_{\text{peak}} \approx 4.45 \text{ N} \), whereas the force decreases to 3.9 N for waveform-III with a low systolic slope. The reason is that the waveform may affect the flow acceleration, and hence influence the net force on the SG wall.

4. Discussion

The specter of migration has been found with all current endografts, including different device configurations and fixation types (Zarins et al., 2003). Severe migration can cause re-pressurization, endoleaks, and hence excessive wall stress, resulting potentially in AAA rupture. Multi-factors, including iliac bifurcation angle, endograft size, blood pressure, endograft wall compliance, iliac branch curvature, and neck length have been reported to influence device migration (Liffman et al., 2001; Mohan et al., 2002; Volodos et al., 2003; Resch et al., 2000; among others). Our simulation results also indicate that iliac bifurcation angle, neck aorta-to-iliac diameter ratio, and SG size influence SG migration. However, we also found that AAA neck angle and blood waveform play important roles in generating a net fluid flow force potentially leading to SG migration. Based on experiments, Resch et al. (2000) tested experimentally the fixation force and found the maximum average fixation forces of 4.5 N for a commercial Talent SG (without hooks) and 9.0 N for Vanguard SG. As demonstrated with our numerical model, actual fixation forces may be inadequate to withstand peak migration forces; for example, in case of a large neck angle (i.e., \( \alpha = 85° \), see Fig. 8), the net downward force was 10 N.

SG drag force is generated by blood pressure and friction on the SG wall. Because the SG is immersed in the cavity’s blood, both sides are exposed to blood pressures. Thus, to obtain the drag force after SG placement in the AAA, the stagnant blood and AAA wall characteristics should be included in the FSI analysis. When considering the SG without combination of the AAA wall and surrounding stagnant blood as done by Morris et al. (2004), we found that the drag force increased by 10–20% upon the sac pressure levels determined by the AAA/SG wall material properties (Gawenda et al., 2004; Li and Kleinstreuer, 2005a, b). The reason is that the sac pressure is directed opposite to the lumen pressure and hence offsets somewhat the drag force calculated by the lumen pressure alone, which leads to a more conservative estimate in SG-anchoring design. As discussed, a large SG size can lead to a higher drag force because the total momentum at the inlet will change with SG size. Nevertheless, based on our calculations pressure is the major contributor to the drag force. A large SG size implies a larger projected surface area onto the transverse plane, and hence a larger drag force. The resulting change in momentum (not including the pressure) only causes a small part of the total drag force (\(< 10%\)), i.e., the reason that a large SG size causes a larger drag force is largely from the impact of the pressure term.

Some limitations should be mentioned. In this model, the AAA wall thickness was selected to be 1.0 mm at the
maximum diameter. However, AAA-wall remodeling might lead to large variations in wall thickness. Iino et al. (2002) declared that no correlations between wall thickness and AAA size were found based on CT-scan images. The average wall thickness for an AAA is probably the same, less or greater than that of the normal healthy aorta. As a result, the AAA wall stress will be reduced. Furthermore, the device slippage is not
considered in this model. This study analyzed wall stresses for smooth, homogeneous and isotropic AAA-SG models by use of linearly elastic materials of zero residual stress. Clearly, patient-specific stented aneurysms exhibit wide variations in shape, size, non-linear material properties, and loading conditions. Even though AAA material is often assumed to be linearly elastic (see Thubrikar et al., 2001; Di Martino et al., 2001; Vorp et al., 1998; Hua and Mower, 2001), Raghavan and Vorp (2000) indicated that hyperelastic material properties might represent AAA walls more accurately.

In this model, the sac blood was assumed to be a stagnant fluid. Clinically, sac blood will thrombose within hours and become solid-like within weeks. Furthermore, there are usually significant amounts of aged thrombus inside the sac. Our previous simulations showed that the existence of a thrombus in the cavity does not affect the drag force measurably, but it can reduce somewhat the AAA–wall stress (Li, 2005). The simplifying assumptions make our results quantitatively limited. Nevertheless, the present analysis introduces a computational FSI methodology for the simultaneous evaluation of blood flow fields, sac pressures, AAA/SG wall stress distributions, and drag forces. Such information is important to provide physical insight into the hemodynamics and biomechanics of stented AAAs.

The following conclusions can be drawn from this analysis:

1. An SG placement can significantly reduce sac pressure, mechanical stress, pulsatile wall motion, and maximum diameter change in AAAs; hence, it may restore normal blood flow and prevent AAA rupture effectively.
2. The SG drag force behaves similarly in trend as the cardiac pressure. Its magnitude depends on multi-factors, including blood flow conditions as well as SG and aneurysm geometries. Specifically, AAA neck angle, iliac bifurcation angle, neck aorta-to-iliac diameter ratio, SG size, and blood waveform play important roles in generating a net fluid flow force potentially leading to SG migration.
3. The drag force can exceed 5 N for an AAA with a large neck or iliac angle, wide aortic neck and narrow iliac arteries, large SG size, and/or abnormal blood waveform. Thus, the fixation of a self-expandable or balloon-expandable SG contact may be inadequate to withstand the forces of pulsatile blood flowing through the implant, i.e., means of extra fixation should be considered.
4. A comprehensive FSI analysis of the coupled SG–AAA dynamics provides physical insight for evaluating the luminal hemodynamics and maximum AAA-stresses as well as biomechanical factors leading potentially to SG migration.

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Further reading