Longitudinal wall shear stress evaluation using centerline projection approach in the numerical simulations of the patient-based carotid artery

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ABSTRACT
In this numerical study, areas of the carotid bifurcation and of a distal stenosis in the internal carotid artery are closely observed to evaluate the patient’s current risks of ischemic stroke. An indicator for the vessel wall defects is the stress exerted by blood on the vessel tissue, typically expressed by the amplitude of the wall shear stress vector (WSS) and its oscillatory shear index. To detect negative shear stresses corresponding with reversal flow, we perform orientation-based shear evaluation. We investigate the longitudinal component of the wall shear vector, where tangential vectors aligned longitudinally with the vessel are necessary. However, resulting from imaging segmentation resolution of patients’ computed tomography angiography scans and stenotic regions, the geometry model’s mesh is non-smooth on its surface areas and the automatically generated tangential vector field is discontinuous and multi-directional, making an interpretation of our orientation-based risk indicators unreliable. We improve the evaluation of longitudinal shear stress by applying the projection of the vessel’s centerline to the surface to construct smooth tangential field aligned longitudinally with the vessel. We validate our approach for the longitudinal WSS component and the corresponding oscillatory index by comparing them to results obtained using automatically generated tangents in both rigid and elastic vessel modeling and to amplitude-based indicators. We present the major benefit of our longitudinal WSS evaluation based on its directionality for the cardiovascular risk assessment, which is the detection of negative WSS indicating persistent reversal or transverse flow. This is impossible in the case of the amplitude-based WSS.

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1. Introduction
The importance of a healthy and functioning cardiovascular system is reflected in the WHO death statistics of 2019. Ischemic stroke was the disease responsible for the highest proportion of deaths across all countries and wealth levels (WHO: The top 10 causes of death 2020). The cause of ischemic stroke is an arterial vascular disease, which in its most common form, atherosclerosis, is an inflammatory response of the vessel wall to lipid metabolism disturbances and endothelial stress. This leads to the formation of multi-focal plaques and thus to the narrowing and hardening of the arteries and consequently to an insufficient supply of oxygen to the brain (Debus et al. 2013). A special role in atherosclerosis development plays the carotid artery, which is responsible for an estimated 18–25% of thromboembolic strokes (Iannuzzi et al. 2021). In the carotid bifurcation the common carotid artery splits into the external and the internal carotid artery. While the former is responsible for supplying blood to the head and upper neck organs, the latter supplies blood to the brain. Both, the death toll of ischemic strokes and the drastic increase in the general prevalence of atherosclerosis, which is related to demographic change and the accompanying burden on health and care system, make it necessary to adequately address the danger posed by atherosclerosis. To provide necessary tools predicting the locations of sites susceptible to atherosclerotic damage, as well as to make recommendations for their optimal treatment, is one of the main goals of modern medicine.

The predictions of atherosclerosis and further cardiovascular risks through numerical simulations have become popular over the last decades. Especially in the field of fluid–structure-interaction there is an inexhaustible range of publications. They span from the incorporation of different mathematical models, such as non-Newtonian fluids (Janela et al. 2010; Hundertmark et al. 2012), different structure models, e.g. shell models and membrane models
(Ciarlet 2000; Čanić et al. 2006), to exploring new numerical methods, which effectively tackle the multi-physicality with splitting techniques (Hundertmark and Lukáčová 2010; Rusnáková et al. 2013; Bukač et al. 2014), just to name a few.

For the purpose of risk quantification, parameters derived from numerical flow data are of great interest, e.g. wall shear stress (WSS) (Taylor et al. 1998; Antiga 2002; Shojima et al. 2004; Lawton et al. 2008; Arzani and Shadden 2016), or oscillatory shear index (OSI) (Antiga 2002; Quarteroni and Gianluigi 2003; Xiang et al. 2011), both referred to be correlated with cardiovascular risk. It has been reported (Ku et al. 1985) that apart from regions with high amplitude WSS, areas with low and temporary oscillating WSS promote atherosclerotic processes. To be more precisely, low mean shear and oscillatory shear stress contribute to an increase in fluid residence time, which can result in the modification of mass transport of atherogenic substances between lumen and wall or interference with endothelial. The multi-directional behavior of the flow induced WSS is thought to play a major role featuring the complex patterns of blood flow and its irregularities, and it has been linked to potential risk zones (Hoogendoorn et al. 2020). In porcine vasculature, it has been observed that sectors exposed to low TAWSS (temporal averages WSS) or to high multi-directional WSS (measured by parameters as OSI, RRT-relative residence time, CFI-cross flow index, or transverse WSS) exhibit significantly higher plaque growth per month than regions with higher TAWSS or lower multi-directional WSS metric levels. Local flow stagnation or low velocity recirculation zones have been found to contribute to thrombus formation (Zilberman-Rudenko et al. 2017), which can then amplify the stenosis or lead to arterial embolism. Concerning the backflow and its link to the lesions of human carotid arteries we refer to the early work of Zarins et al. (1983). Here, carotid bifurcations of asymptomatic patients have been obtained at autopsy. Low shear stresses and negative flow velocities have been measured along the outer wall of the carotid sinus bulb (by laser Doppler anemometry), where lesions in the specimens have been observed. In Hansen et al. (2016), the injurious character of backflow in the descending aorta has been confirmed by applying vector flow imaging. The atherosclerotic plaques were clearly correlated to the systolic backflow on a set of 25 patients. See also the references therein reporting on strong association between the location of systolic backflow and low wall shear stress, and the location of plaque formation. Some of the recent results report on proper visualisation tools for the localisation of cardiovascular risk zones. These are based on the interplay of imaging techniques for exploring the vessel morphology and simulated data as velocity streamlines or WSS, see, e.g. Eulzer et al. (2021) and further citations therein. For those tools and their underlying numerical simulations the patient’s unique vessel morphology plays a crucial role for reliable risk predictions of the above mentioned parameters.

The aim of this study is to reliably quantify the impact of the fluid flow dynamics on endothelial stresses in a clinical patient carotid artery with a topologically complex inner artery surface due to stenotic plaque. The obtained numerical data serve as exploration of potential stroke risks. Our specific goals are:

- Evaluating risk indicators incorporating the multi-directional behaviour of WSS appearing in low and recirculatory flow. These evaluations are sensitive to the orientation of surface tangent vectors. On complex surfaces the generic mesh-based tangents may be inappropriate for this evaluation. We propose an approach based on the construction of properly aligned surface tangents with information on the geometric centerline.
- Demonstrating the feasibility of our approach for implementation of orientation based WSS metrics on complex geometries, its application and the benefit in low and multi-directional shear detection.

The domain of interest is the carotid bifurcation area with its separation of the common carotid artery into the internal carotid artery (ICA) and the external carotid artery (ECA). The 3D lumen of the carotid vessel tree including its stenotic region as well the shape of the surrounding wall tissue have been reconstructed from the computer tomography angiography (CTA) data set of an anonymous, retrospective clinical patient with the method described in Eulzer et al. (2021) and are presented in Figures 1 and 2. This carotid vessel tree as well as its arterial wall shape have been imported as fluid and solid regions in the finite-element-based software Comsol Multiphysics. In the realistic modelling the impact of both, the realistic geometry and the blood rheology may be comparable, as reported in Gambaruto et al. (2011) for the case of cerebral aneurysm. Numerical studies (Janela et al. 2010; Bodnár et al. 2011) address or investigate the effects of blood rheology, considering non-Newtonian shear-thinning or viscoelastic blood models. In addition to shear-thinning particularly significant differences applying quasi-linear viscoelastic Generalized Oldroyd B and nonlinear viscoelastic Giesekus model have been determined in
Romano et al. (2020) in regions with high velocity gradients or in atherosusceptible regions as bifurcations. In the proposed numerical study we focus on evaluation of hemodynamic risk descriptors featuring the multi-directionality of the low shear stress, and we propose an approach of centerline projection for construction of properly aligned tangential field allowing to detect negative values of WSS. We constrict here to the incompressible Newtonian fluid model and give insight into the differences between amplitude based and orientation-based risk descriptors in complex geometries including the compliance of the vessel wall. The impact of the non-Newtonian rheology models on qualitative or quantitative behaviour of studied risk indicators is out of the scope of this study and may be addressed in the future. For the compliance of the vessel wall the fluid–structure interaction (FSI) model describing the interplay of the fluid and thick compliant walls is considered. The corresponding wall tissue mechanics is modeled by the deformation of a linear elastic material.

The obtained numerical velocity data are used to evaluate the hemodynamic risk descriptors: the wall shear stress (WSS) and the oscillatory shear index (OSI), measuring the temporal change of the wall shear stress direction over one cardiac cycle. Herein, we consider the longitudinal component of WSS and compare it to the results for the amplitude of the WSS vector. Moreover, we evaluate the corresponding oscillatory indices using the temporal mean of the longitudinal WSS component, as well as the amplitude of the mean WSS vector. We give their hemodynamic interpretation and discuss the benefits of the orientation-based WSS evaluation in view of reversal flow detection.

For the calculation of the longitudinal WSS value the choice of proper tangential vectors is crucial. The analogous sensitivity of the transversal WSS to the orientation of the tangential field has been addressed in Mohamied et al. (2015, 2017), Gallo et al. (2016), and Hoogendoorn et al. (2020). Regarding the realistic geometries with spatially non-smooth surface topology, the mesh-based erratic tangential vector field leads to problematic, spatially discontinuous behavior of longitudinal (or transversal) WSS on the surface of the extracted geometry. We address this problem and improve the evaluation of the longitudinal component of WSS in complex realistic geometries. Our approach is based on choosing tangential vectors obtained by the projection of the centerline of the vessel tree to the vessel surface, previously used in Morbiducci et al. (2015) or Arzani and Shadden (2016). We present numerical data and two wall parameters WSS and OSI derived from the projection method and from the automatically generated, mesh-based tangential vector fields. Further, we compare the values obtained for rigid as well as compliant carotid vessel walls in order to examine the importance of the compliance of wall tissue in considered mathematical model of the carotid artery.

2. Mathematical modeling of the carotid flow

To investigate the individual risk of arterial defects and imminent health issues, the individual examination of the carotid geometry can give a reliable assessment of the patient’s current situation. We process the CTA scans of a patient to a detailed three-dimensional arterial geometry, as described in Eulzer et al. (2021). The arterial lumen and plaque of the vessel wall are segmented with a semi-automatic labelling approach using 3D Slicer. From the lumen labels, we reconstruct a geometry for the inner vessel wall. The lumen labels are then expanded to approximate the outer vessel wall, from which a second geometry is generated. In sites where hard plaque has been segmented, the
In particular, we denote with \( \Omega_i = \Omega_i^f \cup \Omega_i^s, t \in [0, T] \) the deforming fluid and structural domain in time \( t \), respectively, and their shared boundary with \( \Gamma_{fsi}^i = \Omega_i^f \cap \Omega_i^s \). To model the blood flow in a deforming vessel, the incompressible Navier-Stokes equations in a moving domain are considered in the arbitrary Lagrangian-Eulerian (ALE) formulation. They read as

\[
\rho_f \frac{Du}{Dt} + \rho_f ((u - w) \cdot \nabla)u = \nabla \cdot T_f,
\]

with the Cauchy stress tensor \( T_f = -\rho I + 2\mu D(u) \) and the strain rate tensor \( D(u) = \frac{1}{2} (\nabla u + \nabla u^T) \). The fluid velocity \( u \) and pressure \( p \) are solved in a fluid domain for constant viscosity \( \mu \) and density \( \rho_f \). Here \( w(x,t) = \frac{\partial x(X,t)}{\partial t}, x \in \Omega_i^f \) describes the fluid domain deformation velocity with respect to the reference domain \( \Omega_0^f \) and \( \frac{Du}{Dt} \) is the total (material) time derivative of the fluid velocity.

The boundary of the fluid domain consists of inflow and outflow boundary as well as the shared fluid–structure interface, \( \Gamma_f = \Gamma_{in}^f \cup \Gamma_{out}^f \cup \Gamma_{fsi}^i \). On the fixed inflow boundary \( \Gamma_{in}^f \) a pulsating blood flow rate was implemented, based on measured data taken from (Perktold and Rappitsch 1995) as shown in Figure 6. The outflow was assumed to have zero normal stress (do-nothing condition), that is

\[
T_f \overrightarrow{n}_{out} = -p_{out} \overrightarrow{n}_{out} \equiv 0 \quad \text{on} \ \Gamma_{out}^f. \quad (2)
\]

The mechanics of the arterial wall is modeled by a linear elastic material which reads in the reference configuration as,

\[
\rho_s \frac{\partial^2 d}{\partial t^2} = \nabla \cdot (FS)^T + f_s, \quad \text{in} \ \Omega_0^s,
\]

with \( S = JF^{-1}(C : \varepsilon)F^{-T} \).

Here \( d \) denotes the deformation, \( F = \frac{\partial x}{\partial X}, x \in \Omega_i^s, X \in \Omega_0^s \) the deformation gradient, \( \varepsilon = \text{det} F \) and the second Piola-Kirchhoff tensors is denoted with \( S \). Outer forces acting on the volume are incorporated in \( f_s \). The elasticity tensor \( C = C(E, \nu) \) is given with dependency on the Young’s modulus \( E \) and the Poisson’s ratio \( \nu \). The elastic strain tensor is given by the Green-Lagrange strain \( \varepsilon = \frac{1}{2}(F^TF - I) = \frac{1}{2} (\nabla d + (\nabla d)^T + \nabla d(\nabla d)^T) \). Note, that for small deformation gradients it holds \( S \approx C : \varepsilon \).

The surfaces of the domain clipping, i.e. the annular boundaries of the vessel cuts, surrounding the in- and outflow boundaries of the fluid domain, \( \Gamma_{in}^f \) and \( \Gamma_{out}^f \), are constraint to have 0 deformation. In contrast, the outer surface of the vessel, which would be in contact with surrounding tissue, is able to move freely.

To maintain continuity of velocities and forces at the fluid–structure boundary layer, we enforce the coupling conditions to balance velocities and normal stresses of the fluid and the solid material,

\[
\frac{\partial d}{\partial t} = \overrightarrow{u} = \overrightarrow{w} \quad \text{and} \quad JF_f \overrightarrow{n} = -(FS)^T \overrightarrow{n}, \quad \text{on} \ \Gamma_{fsi}^i. \quad (4)
\]

Here, \( \overrightarrow{u}, \overrightarrow{w} \) stand for fluid quantities transformed to the reference fluid-solid layer.

Note that the model considering rigid walls consists only of the fluid sub-problem (1) defined in the carotid lumen \( \Omega_i^f \), moreover \( \overrightarrow{w} = 0 \) and \( \frac{\partial u}{\partial t} = \frac{\partial w}{\partial t} \). In analogy to the velocity continuity condition in (4) the no-slip condition \( u = 0 \) is prescribed on the vessel wall surface denoted by \( \Gamma_{in}^f \) in case of rigid walls.

### 3. Hemodynamic indicators

#### 3.1. Wall shear stress

The wall shear stress (WSS) measures the endothelial stress exerted by blood on the vessel tissue. To explain the relationship between WSS and zones susceptible to atherosclerosis two main explanatory approaches can be found in the literature. The high shear stress theory identifies sites with prolonged high WSS as risk zones, the low shear stress theory considers also sites with oscillating and low WSS as potentially at risk, the correlation was reported in Ku et al. (1985). For a systematic review of both, see Peiffer et al. (2013).

Principally, the wall shear stress is defined on the interface boundary \( \Gamma_{fsi}^i \) or on the rigid vessel wall \( \Gamma_{in}^f \), as the projection of the normal stress vector \( \overrightarrow{t_f} = -\overrightarrow{F_f} \overrightarrow{n} \) onto the tangential plane,
where $\vec{t}_1$, $\vec{t}_2$ are unit vectors spanning the tangential plane. Alternatively a non-directional quantity describing the amplitude of the wall shear stress vector is frequently evaluated (Quarteroni and Formaggia 2004) as

$$\tau_w^a = ||\vec{\tau}_w|| = \sqrt{(\vec{u}_f \cdot \vec{t}_1)^2 + (\vec{u}_f \cdot \vec{t}_2)^2}.$$  

Different direction-based indicators of WSS have been used to measure the stress exerted by the fluid as well (Antiga 2002; Mohamied et al. 2015, 2017; Gallo et al. 2016; Hoogendoorn et al. 2020). For cylinder-like or other simple geometrical objects vector quantities such as the rotary (transversal) or longitudinal component of the WSS can be considered. In this study, we evaluate the longitudinal component of the wall shear stress vector (longitudinal WSS), aiming to track the backward flow in the carotid artery bifurcation vessel tree, which is defined by (8) as

$$\vec{t}_\ell = \vec{t}_2 \ \text{sign}(\vec{t}_2 \cdot \vec{v}).$$  

The overall flow direction vector $\vec{v}$ has to be specified locally for different sections of the computational domain tree.

Note, that $\vec{t}_\ell$ defined by (8) is revolved from $\vec{v}$ by less then $\pi/2$ and thus aligned almost with the main flow, but it still has the same jumping behavior as the vector $\vec{t}_2$, compare Figure 3 (right). In what follows we present an improved approach for constructing a proper longitudinal tangential field $\vec{t}_\ell$, which is based on the alignment of the carotid tree centerline and can therefore be utilized globally.

3.2. Projection method for tangential field

As depicted above in Figure 3, on complex surfaces the automatically rendered tangent vectors $\vec{t}_2$ do not follow the overall flow direction in some topologically complicated areas. In order to overcome this difficulty we apply the approach based on the knowledge of the centerline of the vessel tree and its projection onto the vessel surface, similarly to the method of Morbiducci et al. (Morbiducci et al. 2015).

At first the centerline is obtained as the set of center-points of the maximally inscribed spheres. Here, we use the 3D Voronoi diagram of the geometry to find and connect the center-points, the method described and implemented in the vascular modeling toolkit (Izzo et al. 2018). The method yields robust and detailed results with a resolution of about 3000 points. After getting the 3D curves of the centerline, its tangential vectors $\vec{c}'$, Figure 4, are obtained from five consecutive points of the curve as an average of two preceding and two subsequent vectors.

Afterwards, the centerline tangent vectors $\vec{c}'$ are projected to the vessel surface, i.e. into each surface point $P_k$. This is done in two steps, first $\vec{c}'$ is extrapolated to the surface points by the geometry tool extrapolate with linear settings in Comsol. Then, the extrapolated centerline tangents $\vec{c}$ are projected into the tangential plane of the carotid artery surface by subtracting its normal component,

$$\vec{t}_\ell = \frac{\vec{c} - (\vec{c} \cdot \vec{n}_f) \vec{n}_f}{||\vec{c} - (\vec{c} \cdot \vec{n}_f) \vec{n}_f||},$$  

here $\vec{n}_f$ are the normal vectors of the carotid surface. The resulting longitudinal tangential field $\vec{t}_\ell$, see
Figure 5, shows a more uniform alignment at first sight compared to the flipped tangential field (8) presented in Figure 3. In this manner, longitudinal tangent vectors \( \vec{t} \) derived from the centerline, with proper unidirectional behavior on the surface, are implemented in Comsol. Both, projected (9) as well as flipped tangents (8) are used for the evaluation of longitudinal WSS and the results are compared in what follows.

3.3. Oscillatory shear index

The oscillatory shear index (OSI) introduced by Ku et al. (1985) is a common indicator for disturbed flow. It characterises the temporal oscillations of WSS through its directional change at any point on the surface in the considered time period. The degree of oscillation is expressed by the ratio of averaged WSS compared to its averaged amplitude over the whole time interval, i.e. in means of temporal mean values. Note, that OSI does not express the frequency of the sign change of the WSS.

We introduce two definitions of OSI, which can be found in literature (see e.g. Taylor et al. 1998; Antiga 2002; Quarteroni and Gianluigi 2003; Quarteroni and Formaggia 2004; Soulsis et al. 2011; Xiang et al. 2011; Blagojević et al. 2013), based either on the amplitude of the temporal mean of the WSS vector, (6), or on the size and sign of the temporal mean of its longitudinal component (7),

\[
\text{OSI} = \frac{1}{2} \left( 1 - \frac{\int_0^T \bar{\tau}_w \ dt}{\int_0^T |\bar{\tau}_w| \ dt} \right),
\]

\[
\text{OSI}^f = \frac{1}{2} \left( 1 - \frac{\int_0^T \tau'_w \ dt}{\int_0^T |\tau'_w| \ dt} \right).
\]

Note, that these formulas differ in the ratio and the norm of the ratio of temporal mean WSS. Consequently, formula (10) defines values of the OSI between 0 and 0.5, where 0 stands for no or a complete change of sign over the mean WSS’s entire time interval and 0.5 for completely balanced sign changes, e.g. oscillations of the mean WSS. Values in between imply corresponding sign
changes of the WSS. In contrast, negative values of the mean longitudinal WSS in (11) play an important role. They lead to a range of OSI values from 0 to 1. Similarly to definition (10), values of 0.5 indicate completely balanced sign changes of WSS as well. An OSI value of 0 represents a point at which the WSS is positive over the entire time interval considered. For the value 1, on the other hand, the WSS is negative over the entire time interval. Values between 0 and 0.5 show predominantly positive, values between 0.5 and 1 describe predominantly negative WSS over the whole time interval. The definition (11) thus allows to locate not only sites of oscillating WSS, but also sites with long-lasting or predominantly negative WSS. Thus, in contrast to (10), definition (11) provides an index that can represent both indicators of low shear stress theory. In what follows, we refer to the directional definition (11) when mentioning the OSI, but we also evaluate the OSI defined with the use of the WSS amplitude (10).

4. Numerical method and convergence study

The numerical simulations have been performed with Comsol Multiphysics (Comsol Multiphysics Reference Manual 2020), Version 5.6 using the MEMS Module to incorporate the interaction of laminar fluid flow with the linear elastic wall material. The software uses the arbitrary Eulerian-Lagrangian formulation, which consists of the Navier-Stokes equations in the Eulerian frame (1) and the solid mechanics equations using the Lagrangian formulation (3). The interaction was chosen to be bidirectional, such that the fluid loading acted on the structure and the wall velocity is transmitted to the fluid. We chose a monolithic, fully-coupled solving scheme to provide a robust solution for the dependent variables consisting of the solid deformation d, the fluid flow velocity u, pressure p and the spatial mesh displacement w.

For the discretization of the fluid velocity and pressure linear and for the solid deformation quadratic finite elements have been chosen. For the time discretization BDF-method of order 1 (for the initialization) continuing with order 2 and an adaptive time-stepping has been applied. The simulations have been performed on a computational mesh consisting of about 163500 tetrahedral elements in the solid domain and 510800 elements in the fluid domain, about 412500 of which are tetrahedral and 98300 are prisms acting as two boundary layers. The whole simulation spans over two cardiac cycles, i.e. 1.8s, and is driven by a pulsatile flow rate presented in Figure 6, starting with the domain at rest, i.e. zero deformation and zero flow at t = 0. All results presented in the next section are chosen from the second cycle where the flow is fully developed.

4.1. Convergence study

We perform mesh convergence study to demonstrate the convergence of our numerical solution towards a highly resolved reference solution. For this purpose the fluid flow problem with solid vessel walls was solved only to reduce the computational costs. The spatial mesh error was computed using a set of eight meshes, approximately doubling the mesh element number from mesh i to mesh i + 1. To compare numerical solutions on different meshes with non-coinciding mesh nodes, the linear shape functions (P1 finite elements) are applied. The solution difference is realised with use of join solution feature in Comsol by projection of the lower-mesh solution onto the higher mesh. The spatial discretization error has been evaluated for fluid velocities and for the longitudinal WSS in means of weighted L²-norm of the difference of the reference and actual i-th mesh solution obtained on meshes no. 1–6,

\[
\text{err}(u_i) := \frac{1}{\sqrt{|\Omega|}} \|u_i - u_{ref}\|_{L^2(\Omega)} \quad \text{and} \quad \text{err}(\tau_i) := \frac{1}{\sqrt{|\Gamma_w^i|}} \|\tau_i - \tau_{w,ref}\|_{L^2(\Gamma_w^i)} \quad i = 1, \ldots, 6,
\]

(12)

in the time-point of the maximal flow of the second cardiac cycle, t = 1.1s. Here, \(u_i, \tau_w^i\) are numerical data obtained on the i-th mesh. The reference solution has been obtained on mesh no. 8 consisting of a total of 8 717 584 tetrahedra and prism elements. The mesh errors (12) have been evaluated in the chosen region of our computational mesh identical to geometrical region of numerical results for WSS und OSI presented in Figures 10–16.

The mesh element sizes, measured by the diameter the maximum inscribed sphere, have been averaged for each mesh and spatial errors have been related to the mean mesh sizes \(h_i\). The descending sequence of \(h_i\) starting with \(h_1 = 1.054 \text{ mm}\) is presented in Table 1, whereby the decrease factor

\[
a_i := h_i / h_{i+1}
\]

lies between 1.271 and 1.22. In Figure 7, the errors with respect to \(h_i\) are presented in logarithmic scale. For the decreasing error curves one can observe a slope of approximately one, which slightly differs for higher and lower mesh sizes. In analogy to the error
estimation for finite element method with a uniform mesh and constant element sizes $h_i$, $||u_i - u_{\text{exact}}||_2 \approx C h^p$, $C < \infty$, where the convergence order $p$ is identified with the slope of the logarithmic error curves for different $h$, we denote the slope of our logarithmic error curve by the experimental order of convergence (EOC). EOC can be obtained by comparing errors of two consecutive meshes,

$$EOC(u_i) = \frac{\log_{10}(\text{err}(u_i)) - \log_{10}(\text{err}(u_{i+1}))}{\log_{10}(h_i) - \log_{10}(h_{i+1})} = \log_{10}\left(\frac{\text{err}(u_i)}{\text{err}(u_{i+1})}\right),$$

(13)

analogously for $(\tau'_{w})_i$, $i = 1, \ldots 6$.

The results of our mesh convergence study are summarised in Table 1 and show good convergence of the numerical velocities as well as for longitudinal wall shear stresses to the reference numerical solution. The EOC, initially lower than one, increases continuously with decreasing mesh size, with a small deviation in case of $(\tau'_{w})_3$. The averaged EOC for the velocities is about 1.13 and for the longitudinal WSS it is about 1.21, altogether a super-linear averaged convergence to the reference solution is obtained in our simulations with rigid vessel walls. Applying the finite element method with linear P1 elements, second order convergence rate is expected for spatial error. The decreased experimental order of convergence is caused by the discontinuity of the boundary conditions for velocity on the edges common for the inflow $\Gamma_{\text{in}}$ as well as vessel wall boundary $\Gamma_{\text{w}}$, where the no-slip boundary condition meets the non-zero inflow velocity, considered to be constant over $\Gamma_{\text{in}}$.

As a trade-off between numerical error behaviour and the computational time our results presented in Section 5 have been computed on a mesh consisting of about 510800 elements in the fluid domain, as mentioned above. Since this mesh is close to the mesh no. 4 from Table 1, we can quantify the velocity error to about 0.1 m/s, which is 9.7% of the maximal systolic velocity and the longitudinal WSS $(\tau_{w}^l)$ error about 1.7 N/m², i.e. about 6% of maximal $(\tau_{w}^l)$ in the presented results.

### 4.2. Impact of the boundary layer in the WSS evaluation

Since the wall shear stress is dependent on velocity gradients close to the vessel wall, the resolution of the boundary layer plays an important role in the WSS evaluation and may influence the WSS surface distribution. As described above, in our simulations we consider a computational mesh similar to mesh no. 4 with two mesh boundary layers in a thin region close to the vessel wall. To investigate the impact of the boundary layer resolution we evaluate the change of the longitudinal WSS $\tau_{w}^l$ as well as WSS amplitude $\tau_{w}^a$ with respect to two additional refinements of the thin boundary layer, i.e. we compare results for 2, 4 and 8 mesh layers in the thin boundary region. The detailed results for $\tau_{w}^l$ in an exemplary region of the carotis communis prior to bifurcation during systole are presented in Figure 8. Here, the thickness of the boundary layer was approximately 0.23–0.3 mm, i.e. 7–9% of the radius when considering idealized circular cross sections. No significant qualitative difference in the surface distribution of $\tau_{w}^l$ can be ascertained in the upper row of Figure 8; however, one can observe slightly higher maximal WSS values for higher resolutions of the thin boundary layer. The relative difference of $\tau_{w}^l$ obtained for coarser and finer boundary layer resolution amounts up to 15% comparing the original mesh with 2 and the mesh with 4 mesh boundary layers. This difference decreases to maximal

**Table 1.** Spatial discretization errors related to mean mesh size.

| Mesh (i) | # of elements | $h_i$ (mm) | $\text{err}(u_i)$ (m/s) | EOC ($u_i$) | $\text{err}(\tau_{l})$ (N/m²) | EOC ($\tau_{l}$) |
|----------|---------------|------------|------------------------|------------|-------------------------------|----------------|
| 1        | $6.106 \times 10^4$ | 1.054 | 0.1689 | 0.613 | 3.046 | 0.726 |
| 2        | $1.250 \times 10^5$ | 0.829 | 0.1458 | 0.736 | 2.559 | 0.896 |
| 3        | $2.484 \times 10^5$ | 0.667 | 0.1241 | 0.956 | 2.104 | 0.788 |
| 4        | $4.584 \times 10^5$ | 0.544 | 0.1021 | 1.497 | 1.792 | 1.480 |
| 5        | $1.049 \times 10^6$ | 0.429 | 0.0716 | 1.819 | 1.262 | 2.179 |
| 6        | $2.079 \times 10^6$ | 0.351 | 0.0497 | – | 0.814 | – |

*Figure 7. Decrease of $L^2$ error norm (12) for $u$ and $\tau'_{w}$ with respect to the mean mesh size, gray dashed lines with slopes 1 and 2 represent first and second convergence order.*
9% when comparing the mesh with 4 and 8 mesh boundary layers, cf. Figure 8, bottom. Similar relative error of maximal 16% and 9% have been obtained for \( \tau_w \) when comparing meshes with 2, 4 and with 4, 8 boundary layers, respectively, in this region of the carotis communis prior to bifurcation (not presented here).

In the whole considered area of interest of the convergence study (presented in Figures 10–16) we have obtained a difference in weighted \( L^2 \)-norm (12) of 0.157 and 0.093 N/m² for \( \tau_w' \) and 0.485 and 0.380 N/m² for \( \tau_w'' \) for two subsequent refinements of the thin boundary layer. In the relation to the weighted \( L^2 \) discretization error presented in Table 1, which is 1.7 N/m² for \( \tau_w' \) and mesh no. 4, this weighted \( L^2 \)-difference of longitudinal WSS caused by the additional resolution of the boundary layer amounts only 9.2% and 5.5% of the overall \( L^2 \)-error of \( \tau_w' \) (1.7 N/m²) for one and two additional refinements, respectively. Applying the metric triangle inequality, by addition of these two errors the overall difference of \( \tau_w' \) induced by boundary layer refinements amounts not more than 14.7% of the discretization error, i.e. 0.25 N/m². In view of this relatively small quantitative difference of WSS and its not substantial surface distribution change the risk indications focusing on WSS surface distribution and its temporal and directional changes are not significantly affected by the resolution of the boundary layer in our study.

5. Results and discussion

The evaluations of numerical data and considered wall parameters are compared for four model configurations. In two different vessel wall models: rigid & elastic vessel walls two different tangential fields \( T_{\ell}^f \) : flipped (8) & projected tangential field (9) have been used to calculate the longitudinal WSS (7) and the corresponding OSI (11), the overview of model configurations can be found in Table 2. Additionally, the amplitude-based wall parameters (6), (10) are evaluated as well and compared to its directional counterparts.

In every simulation fluid density \( \rho_f = 1000 \text{ kg m}^{-3} \) and constant viscosity \( \mu = 0.00345 \text{ Pa s} \) was chosen. The wall parameters density \( \rho_s = 1070 \text{ kg m}^{-3} \), Young’s modulus \( E = 0.5 \text{ MPa} \) and Poisson’s ratio \( \nu = 0.17 \) \( \nu \in [0.17, 0.5] \) were used in simulations (c) and (d) with fluid–structure interaction.

In Figure 9, the blood velocity streamlines in the stenotic region of the ICA are presented at the time

![Figure 8](image)

**Figure 8.** Upper row: longitudinal WSS \( \tau_w' \) evaluated on mesh no. 4 with 2 (left), 4 (middle) and 8 (right) mesh boundary layers. Bottom: relative error with respect to the maximal WSS, left: for 2 and 4 boundary layers, right: 4 and 8 boundary layers. Color bars are supplemented with minimal/maximal surface values on their bottom/top.
of maximal flow-rate, $t = 1.1s$. The observed vortices are located around the stenotic bulges in the area adjacent to the stenosis and are more conspicuous in the rigid vessel. Note, that the appearance of vortices and backward flow is related to high OSI$^l$ values, observed in Figure 15, and may imply progression of lesions along the carotid artery tree, which is a common hemodynamic hypothesis (Spanos et al. 2017). Note that amplitude-based OSI presented in Figure 16 only indicates the edges of these areas. On the other hand, high-valued and unidirectional velocity streamlines are observed along the inner wall of the ICA and are related to high longitudinal WSS values $\tau_w^l$, as well as the amplitude $\tau_w^a$ of WSS vector, observed in Figures 10–13. The latter fits to the velocity profile observations in Zarins et al. (1983).

### 5.1. Wall shear stress

In what follows we present the wall shear stress computed from numerical data in means of its longitudinal component, $\tau_w^l$, (7), as well as the non-directional quantity $\tau_w^a$, (6) measured by the amplitude of the WSS vector. To compare the WSS distributions for compliant and rigid walls the results are presented on the inner arterial wall in the reference geometry frame.

#### 5.1.1. Longitudinal WSS

The longitudinal WSS (7) is evaluated on the carotid surface as well as along chosen surface curves and compared for model configurations (a)–(d), see Table 2.

| Configuration | Properties                  |
|---------------|-----------------------------|
| (a)           | Rigid walls/flipped tangents|
| (b)           | Rigid walls/projected tangents|
| (c)           | Elastic walls/flipped tangents|
| (d)           | Elastic walls/projected tangents|

In Figure 10, the surface distributions of $\tau_w^l$ for the four configurations at the time of highest flow-rate are presented. All plots (a)-(d) show high positive WSS up to $35 N/m$ in the bifurcation and in the sinusoidal constrications of the stenotic bulge of the ICA. The center of the bifurcation and the regions around the stenotic bulges of the ICA are regions of low and negative WSS. In configuration (a), local point-wise extreme values of $\tau_w^l$ appear using the flipped tangent vectors on rigid surface. These are smoothed out in (c) on the deformed surface with elastic walls. Besides these very local phenomena in (a), wider areas of negative extreme values occur close to the separation point of the bifurcation in configurations (b) and (c), compared to (d). The occurrence of these extreme values is associated with the alignment of chosen tangential fields, which differ for the considered model configurations at the bifurcation point. We demonstrate this coherence for automatically rendered flipped (c) and projected tangents (d) on compliant walls in Figure 11. Indeed, a clear side separation of the flipped tangent vectors losing their longitudinal alignment even before the bifurcation can be observed in plot (c), explaining the discontinuity and the appearance of negative WSS values up to $20 N/m$ in this area. On the other hand, the longitudinal continuance of projected tangents until the separation point is apparent in plot (d). Obviously, configurations with flipped tangent vectors lead to a spurious longitudinal WSS evaluation on the carotid surface close to their bifurcation point, whereas the tangent field (d), which is projected from the centerline, seems to appropriately map the main flow and its separation in this problematic area.

To get more detailed comparisons, $\tau_w^a$ along the chosen surface curves is shown in Figure 12 for configurations (b), (c), (d). Its values in configurations (b) and (d) are almost identical and differ at most by $1 N/m$ along the circumferential curve (1). On the longitudinal line (2) we observe a very good agreement of (b) and (d). The situation is slightly different in case (c) with flipped tangents, where local deflections of WSS values can be observed. These are caused by the previously discussed alignment of differently flipped tangent vectors presented in Figure 11. The negative deflects of $\tau_w^a$ along the longitudinal curve (2) are not visible in Figure 11 due to a different viewpoint, nevertheless they can be identified with local blue areas on the outer wall of ICA in plot (c) of Figure 10.

Generally, we can conclude that the choice of the tangential field has a considerable effect on the evaluation of longitudinal WSS in the bifurcation region of the carotid artery surface, with the projected tangent vectors being the most suitable for this analysis. After passing the problematic area of the bifurcation the WSS results for projected and flipped tangent fields are comparable at maximal systolic flow, whereas certain inaccuracies occur using automatic flipped tangent vectors. This is because of the misalignment in longitudinal direction on complex and uneven surfaces. In addition, effects of the wall movement in FSI models, are present around the bifurcation point, compare e.g. plot (b) for rigid and (d) for compliant walls in Figure 10.

#### 5.1.2. Vector-valued WSS

The WSS on the carotid inner surface measured by the amplitude of the WSS vector $\tau_w^a$ (6) is presented
in Figure 13 with corresponding arrows of vector $\mathbf{\tau}_w$ as well as its longitudinal components $\mathbf{\tau}_w'$ at the time point of maximal flow in the reference geometry frame. One can observe similar surface distribution of the WSS amplitude and of the size of its longitudinal component comparing Figures 11(d) and 13. Let us note, that the opposite direction of $\mathbf{\tau}_w$ with respect to the main flow, which occurs sometimes in blue areas with low WSS amplitude, cannot be recognised by $\mathbf{\tau}_w'$. Thus the backward flow cannot be detected in the surface distribution in Figure 13. However, this low WSS amplitude are predestined for complex, multi-directional flow patterns as backward and transverse flow or vortices. The opposite direction of $\mathbf{\tau}_w'$ can be tracked by its negative longitudinal component $\mathbf{\tau}_w''$. We demonstrate the feasibility of our longitudinal WSS evaluation approach for the detection of complex, backward and transverse flow in view of surface areas of negative $\mathbf{\tau}_w'$ in Figure 14. The negative sign and the sign change of WSS resulting from opposite alignment with respect to the main flow plays also a role in the evaluation of the oscillatory flow behavior, and it is discussed in what follows.

5.2. Oscillatory shear index

In this section we present the temporal change of both, the longitudinal WSS and the vector-valued WSS during the whole cardiac cycle in means of the oscillatory shear index and compare them for compliant as well as rigid wall models. The results are presented on the inner arterial surface in the reference, i.e. the initial geometry frame.

At first, the oscillatory index (11) for the longitudinal WSS (7) is presented for the fixed wall model using projected tangents (b) and for the FSI model using flipped (c) and projected tangents (d) in Figure 15. Comparing the results, almost no difference in the OSI evaluation for configurations (b) and (d) with projected tangents can be observed. The only region of difference worth mentioning spreads out in the bifurcation area, where the centerline tangents have been projected on different surfaces, obviously due to the wall deformation in case (d). In contrast to (b) and (d), configuration (c) shows many small-scale and point-like extreme values, which is a consequence of the discontinuity and deflections of WSS arising from the erratic alignment of the flipped tangents in some surface regions discussed above.

Concerning the hemodynamical interpretation of the results presented in Figure 15, conspicuous red regions of maxima indicating long-lasting negative WSS inside and oscillating WSS at their edges (green transition zones) can be observed in the bifurcation zone as well as prior and posterior to the sinusoidal stenotic occlusion of the ICA in all model configurations. Further punctual abnormalities are present, e.g. after the stenotic occlusion on the left side of the ICA wall. In consistency with the streamlines presented in

Figure 9. Velocity streamlines in the stenotic region of ICA (stenotic plaque marked with arrows) for both vessel wall models colored by velocity magnitude, $t = 1.1$ s, viewpoints from front and back.
Figure 9 and their link to negative longitudinal WSS, Figure 14, red OSI regions are related to backflow and vortices adjacent to the stenosis bulges of the ICA or prior to the bifurcation. Those maximum regions are an indicator of insistent reversal flow (vortices) and the green transition zones of high longitudinal WSS oscillations indicate the pathological progression of mechanical damage of the artery wall, according to the hemodynamic hypothesis.

Finally, a second oscillatory shear index (10) based on the amplitude of the vector-valued WSS computed using the compliant wall model is presented in Figure 16. Here, the red maximum values correspond to WSS regions with zero temporal mean and represent zones of complete sign balance of $\tau_w$, indicating temporal oscillations. As mentioned in Section 3.3, static vortices and continuous recirculations, corresponding to a permanent negative sign of the WSS, are zero valued in this OSI definition and cannot be tracked here. Nevertheless, the high OSI edge-like regions in Figure 16 are in good consistency with the green transition zones observed in Figure 15 – (b),(d), both indicating high wall shear stress oscillations, and thus possible pathological progression, e.g. on the right and back side of the common carotid artery prior to the bifurcation point, or on the upper back side of the considered ICA region. Small discrepancies between the maxima edges of OSI and the green transition zones of OSI in Figures 15
Figure 11. Surface distribution of longitudinal WSS evaluated using flipped (c) and projected (d) tangent fields (shown as normalized arrows), evaluation with compliant walls at $t = 1.1$ s, viewpoint from back (different viewpoint as in Figure 10).

Figure 12. Upper: circumferential (1) and longitudinal (2) intersection curves for the evaluation of WSS. The arc length of (1) starts at the respective green point and follow a clockwise direction. Below: comparison of longitudinal WSS for configurations (b)–(d) along intersection curves (1),(2).
Figure 13. Surface: $\tau^w_s$ – amplitude of the WSS vector on compliant walls at $t = 1.1$ s. Arrows: WSS vector $\tau^w$ (black) and its longitudinal component $\tau^w_l$ (red), both proportional to its size.

Figure 14. Low velocity streamlines and areas of negative $\tau^w_l$ in systolic (left and middle) and diastolic (right) time points demonstrate the link between the negative longitudinal WSS (coloured surface areas) and the oppose or disturbed flow in low shear sites. The velocity streamlines presented also in Figure 9 (right) are filtered up to 0.4 m/s (systole) and up to 0.14 m/s (diastole). The WSS-vectors $\tau^w_s$ in areas of its negative longitudinal component are proportional to their amplitude.
and 16 are obviously caused by differences in the considered wall shear stress indicators $\tau_w$ and $\tau'_w$ in used OSI definitions.

6. Conclusion

In this contribution, a computational study of fluid dynamic and hemodynamic risk parameters has been performed for a carotid artery. The patient-based lumen and its surrounding walls have been imported into the numerical software Comsol Multiphysics and were supplemented for one with fluid dynamics confined by rigid walls as well as a FSI model for compliant artery using the linear elasticity deformation model. The effects of fluid stresses on the arterial wall have been quantified using established hemodynamic risk factors: wall shear stress and oscillatory shear index. Following the low shear theory, we focused on exploration of low and negative WSS and evaluated the longitudinal component of WSS vector (longitudinal WSS) allowing to track the reverse flow in patient-based morphology. The presented results demonstrate the strong dependency of the orientation-based longitudinal WSS and its OSI-index on the proper construction of tangential vectors and
address this problem on topologically complex surfaces obtained from patient CTA scans. For the studied carotid artery tree, we applied the projection of centerline tangents to the inner arterial surface in order to obtain properly aligned and smooth tangential field, and compared the longitudinal WSS computed for projected as well as generic mesh-based tangent vectors. Since the projected tangential field retains the longitudinal alignment on the craggy surface and maps the flow separation in the bifurcation area much better than the automatically generated tangent vectors, reliable numerical results for longitudinal WSS, allowing hemodynamic predictions of reverse flow have been obtained by applying the projection method.

For comparisons, the commonly used vector-valued WSS and its amplitude have been evaluated as well and its oscillatory behavior has been quantified by the corresponding OSI index. Since the WSS-vector amplitude and the amplitude of its temporal mean does not track its opposing orientation, reversal flow or vortices cannot be detected, which is confirmed by our numerical results. Even though the OSI index, based on the vector-valued WSS instead of its longitudinal component, indicates flow oscillatory regions well, it is not able to detect insistent reversal flow regions characterised by low and negative WSS. Hence, the major benefit of our modeling approach using the direction-based longitudinal WSS and its oscillatory index lies the investigation of sites of low shear stresses and persistent reversal flow.

Our study presents an approach for numerical evaluation of directional change of shear stress metrics in realistic vessel anatomy to assess the potential stroke risk for a patient. However, several improvements in the modelling are possible. At first, instead of flow waveform from literature, patient-specific flow-rates or pressure wave-forms in means of boundary conditions should be complemented. Alternatively, on the daughter branches outflow boundary condition taking into account the flow resistance of the subsequent part of the circulatory system may be considered, see (Vignon-Clementel et al. 2006). Secondly, more accurate non-Newtonian rheology models including shear-thinning and preferably nonlinear viscoelasticity of the blood, e.g. as used in Romano et al. (2020) may improve the prediction of hemodynamics and of the wall shear stress descriptors in further investigations. In particular, the impact of non-Newtonian modelling in the detection of multi-directional low shear flow regimes can be of interest. Finally, the range of risk prediction tools can also be extended by considering further multi-directional WSS parameters, where the choice of the tangential field is of particular importance. According to Hoogendoorn et al. (2020), time-averaged WSS (TAWS) (Arzani and Shadden 2016; John et al. 2017) and relative residence time (RRT) (Soulis et al. 2011; Gorring et al. 2015; Hashemi et al. 2021) are strong predictive clinical markers for disease development, even in early stages of atherosclerosis. Moreover, transversal WSS (Piefier et al. 2013; Pedrige et al. 2015; Mohamied et al. 2017) and cross flow index (CFI) (Mohamied et al. 2017) have shown a predictive value when complex fluid flow appears in later phases of atherosclerosis. Taking those parameters into account, a more differentiated prediction of atherosclerotic development can be achieved.

**Disclosure statement**

No potential conflict of interest was reported by the authors.

**Ethical statement**

The principles of the Declaration of Helsinki and the requirements of medical ethics and confidentiality have been respected in this study. Only non-identifiable, retrospective CTA scans have been processed for the computational geometry. The German General Data Protection Regulation does not concern the processing of anonymous information, including statistical or research purposes, see https://dsgvo-gesetz.de/erwaegungsgruende/nr-26/.

**Note**

1. The choice of Poisson’s ratio has negligible impact on the vessel wall displacement in our modeling.

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