

Article

Blood Flow Analysis and Structural Analysis of Arterial Wall in a 3D Model of the Carotid Artery Based on Magnetic Resonance Imaging

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Abstract

The analysis of blood flow behavior has been studied in various aspects. Recently, these studies took a turn when they were developed using Computational Fluid Dynamics (CFD), thereby advancing understanding of fluid behavior. Artery geometries and the properties of flow offer a field of interest for developing studies in which the behavior of liquid and structures can be observed. Biomodel development has evolved to allow for greater ease and precision. With a geometry similar to the real one, mechanical properties can be used to conduct studies with greater precision, yielding greater results on the behavior of a fluid and a structure. This research focuses on developing a biomodel of the right carotid artery. CFD and structural analyses are performed using the generated arterial geometry and a control volume that simulates blood flow. The structural analysis was performed using the Finite Element Method (FEM) to obtain results that provide data on the effects on the arterial walls. The results presented focus on the velocity, pressure, and wall shear stress generated by the flow. In addition, a section on displacement and Von Mises stress results is presented, demonstrating how the structure of the arterial walls is affected by these effects.

Keywords: arterial wall analysis; blood flow; biomodel; computational fluid dynamics; one-way sequential stress



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1. Introduction

The cardiovascular system consists of a complex network responsible for transporting and regulating blood flow through a pulsating and continuous flow. From a biomechanical perspective, the system can be defined as a closed hydraulic circuit. It consists of a natural pump that drives the flow (the heart) and a network of vessels that allow for deformation due to their mechanical properties. The blood pumped by the heart is propelled by the left ventricle, which generates sufficient pressure to distribute it throughout the body. The

cardiac cycle is divided into two phases: systole, the ejection phase during which pressure increases, and diastole, the phase during which pressure decreases and the ventricle fills with blood [1,2].

Arteries are a fundamental part of the cardiovascular system because they transport oxygenated blood throughout the body (Figure 1). Arteries are blood vessels that can be compromised by various factors that affect both the arterial walls and blood flow. Currently, arteries are studied in various ways to understand their behavior in a healthy state and in the presence of any pathology that could compromise their structure [1,3]. Arteries are often classified as complex due to their three-layered structure. The integrity of these layers helps regulate the pressure generated within the walls. Frictional forces exerted on the wall can remodel the structure, affecting the composition of the endothelial layer—the innermost layer of the arterial wall and blood flow. Flow disturbances can lead to changes, such as recirculation, or turbulence [4]. These types of changes are often associated with the development of pathologies. Currently, the field of biomechanics studies research the interaction between flow and structure using computational Fluid–Structure Interaction (FSI) models [5]. However, when the primary focus is evaluating structural vulnerability, one-way sequential stress analysis offers a robust and computationally efficient alternative.

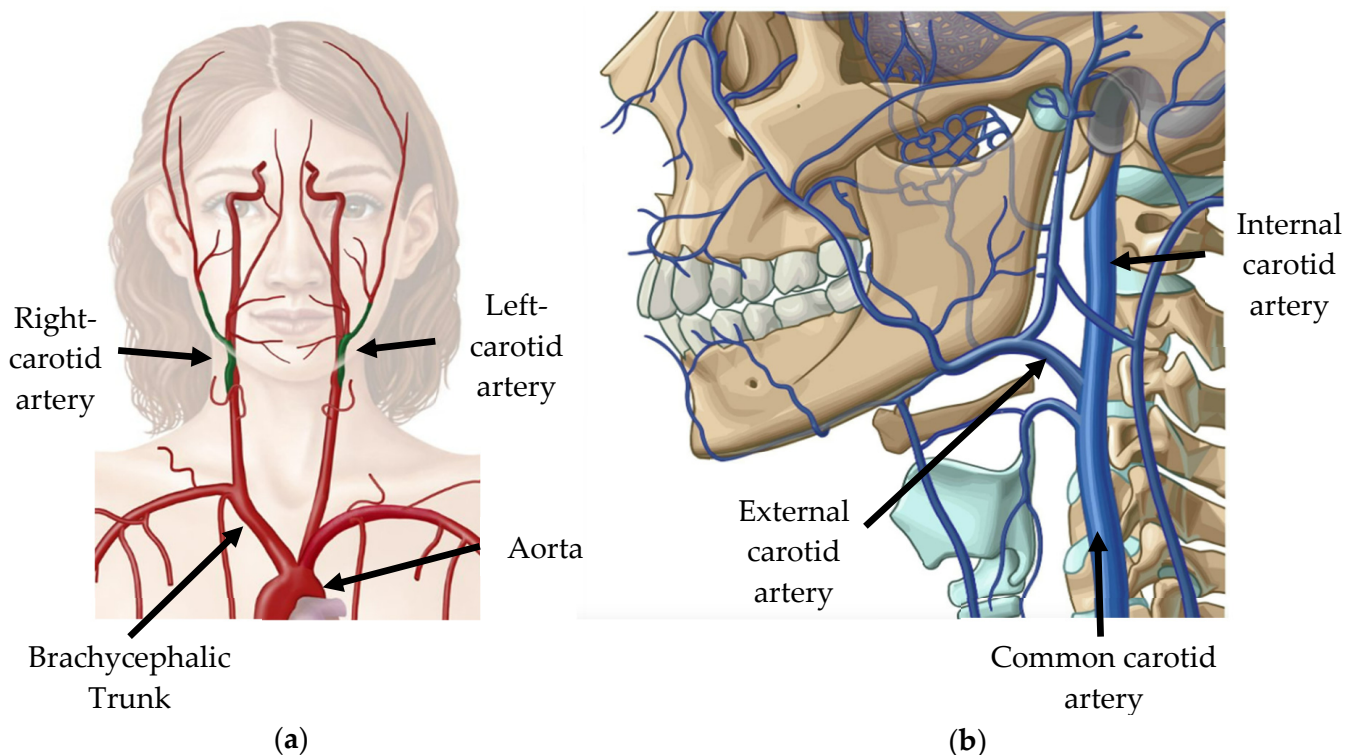


Figure 1. Arteries and carotid artery. (a) Arteries of the head and neck. (b) Segments of the carotid artery.

One way to see how they are affected is through imaging. Imaging focuses on the production and analysis of images of the human body, looking inside it in order to diagnose, predict, and treat diseases. Capturing morphological and functional data from soft tissue is different and, in some ways, more complicated. Various methods are used to achieve higher image resolution in soft tissue imaging, such as of arteries. These images can be produced using Computed Axial Tomography (CAT), Magnetic Resonance Imaging (MRI), and Ultrasound [6]. Each of these methods offers advantages for imaging soft tissue, and they can be distinguished by how they affect the patient or by the resolution they provide for arteries, allowing clearer differentiation of their geometries. To visualize the arteries

correctly with CTA, an angiogram is performed, which uses X-rays and an injected contrast medium to visualize the arteries. This can be done using a CAT scan or an MRI, both of which offer advantages depending on what is required. One advantage of MRI angiography is that it offers better contrast in soft tissues, such as arteries [7]. However, MRI can be categorized into three approaches for visualizing arteries. One of these methods provides an evaluation without contrast material, but the scan time can be lengthy. The other two methods allow the use of contrast material, differing primarily in what the images are intended to show: one allows quantification of blood flow volume, while the other provides better visualization of the vessel walls with a shorter scan time [8]. Another method for obtaining images is Doppler ultrasound, which allows one to observe the direction and presence of blood flow through the vessels and provides a clearer view of the composition of the arterial wall structure. However, this imaging technique relies on video rather than still images, which can complicate the process of reconstructing a 3D model [9].

Based on the images obtained with the imaging technique, they can be used to reconstruct the anatomical structure. The reconstruction of biomodels has been used to perform various types of analyses according to the conditions found in the human body. The way the reconstruction is performed may vary, as there are different approaches [10]. It can be developed using CAD (Computer-Aided Design) or software that supports image segmentation to reconstruct the geometry. The development of these types of models allows for manipulation to perform tests in various ways [11]. One of these has been the analysis of the artery's hemodynamics and how its structure is affected. In this case study, a healthy carotid artery was created in a 3D model to develop sequential CFD and structural analysis. The biomodel was developed from magnetic resonance angiography images. Physiologically, the pressure pulses generated by blood flow exert mechanical loads that deform the arterial walls [12]. Characterizing the structural response between blood and arterial walls is important for the understanding of how pathologies develop in areas where geometric changes occur, such as at the bifurcation of the carotid artery. These changes can lead to turbulent flow or recirculation, which can compromise structural integrity.

2. Materials and Methods

The images on which the artery reconstruction was based are of the right carotid artery. These images allow the model to be developed from the point of origin, which is the brachiocephalic trunk. However, the research focuses mainly on the bifurcation between the common carotid artery (CCA), the internal carotid artery (ICA), and the external carotid artery (ECA). The images shown are axial, coronal, and sagittal views of the human body. Each view includes a segment showing the carotid artery contours [13] (Figure 2). The images were obtained through the IMAIOS website, which offers a wide variety of MRI images of various systems of the human body. To access all the images and other features, a monthly subscription fee is required [13]. Eighty images from the axial plane, 20 images from the coronal plane, and 22 images from the sagittal plane were used. Based on these images, a 3D model of the carotid artery was reconstructed using image segmentation within CAD software SolidWorks 2020.

The reconstruction of the artery within the CAD software was based on superimposition planes onto which the magnetic resonance images were placed [14]. The complete geometry extends 157 mm in its length, measured from its point of origin at the brachiocephalic trunk. The point of interest in the investigation is the bifurcation found in the artery; simulating the entire length was computationally inefficient and unnecessary for the scope of this study. Consequently, a section cut was made in the geometry, focusing on the CCA, ICA, and ECA, leaving an extension of 87 mm for the carotid artery geometry (Figure 3). The contours of the artery were traced, and its lumen was measured. Because

it has different circumferences, it is assumed that the lumen will vary with the geometry, so it is considered to be between 4.5 and 7 mm, according to the findings of the study by Krejza et al. [15]. The wall thickness was determined from the magnetic resonance images. However, based on the clinical and mechanical literature, a thickness range of 0.6 to 0.9 mm was determined, this is based on the findings described by Barrat et al. in their research [16] (Figure 4).

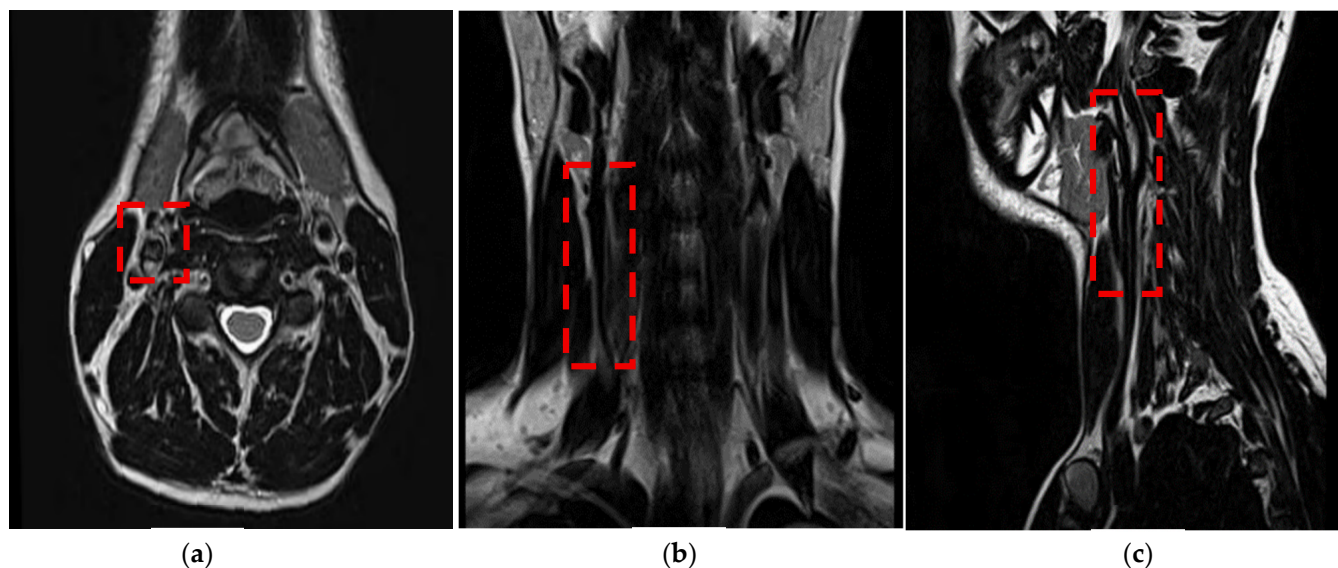


Figure 2. Artery seen from different planes. (a) Axial plane. (b) Coronal plane. (c) Sagittal plane. The red box shows the location of the carotid artery as viewed from different planes.

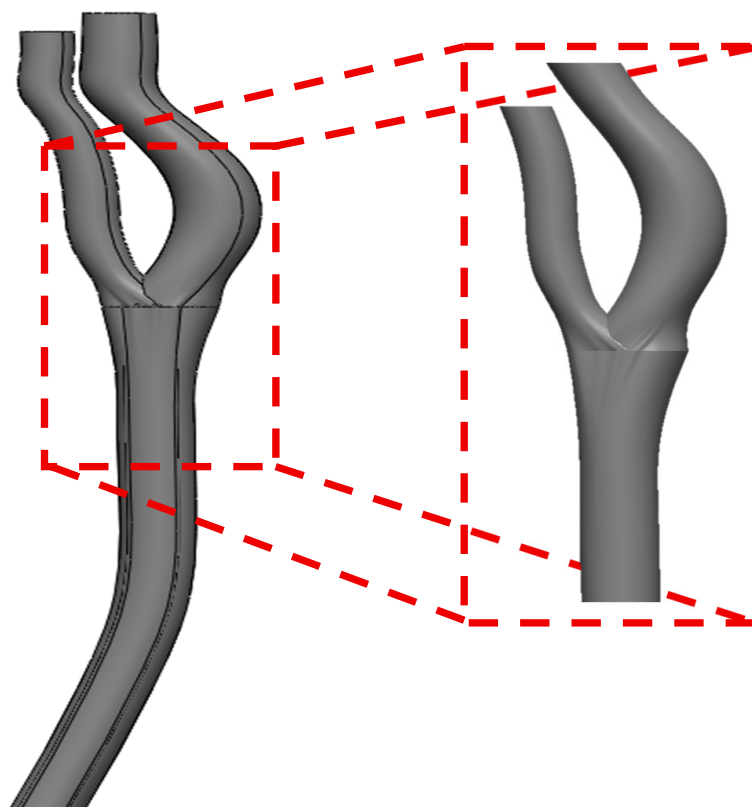


Figure 3. Carotid artery model with section cut for analysis.

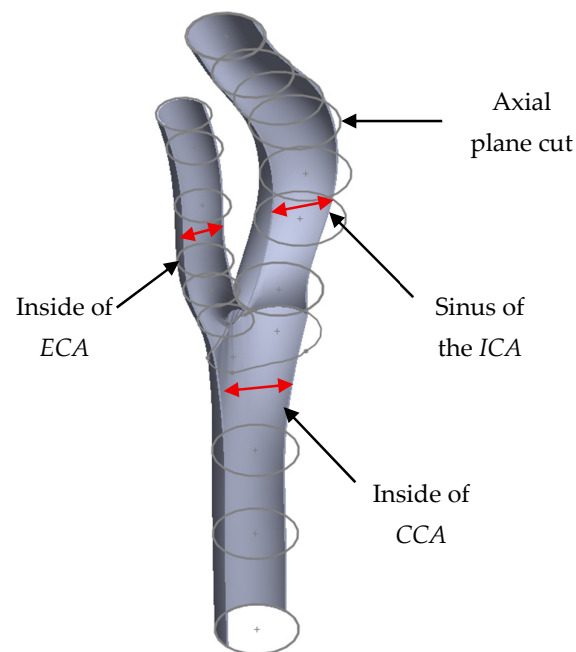


Figure 4. Inside the carotid artery model. The red arrows indicate the internal opening of the geometry, which simulates the lumen of the artery.

3. Results

3.1. Numerical Method

Computational Fluid Dynamics (CFD) were performed using ANSYS Fluent 2021 R2. Fluid analyses can be carried out depending on the conditions being handled. These can vary depending on whether it is an open or closed system, the properties of the flow, and the behavior that it may exhibit. In Fluent, a control volume can be generated for each case (Figure 5) [17]. As the analysis focuses on flow behavior in the carotid artery, a control volume is generated based on the provided geometry. This control volume will have one inlet through the CCA and two outlets: one through the ICA and another through the ECA. The Fluent section of ANSYS is based on the Navier–Stokes equations for the assessment and prediction of flow behavior. Implementing the governing equations requires precise definitions of the boundary conditions (Figure 5) [18]. This approach ensures conservation of mass and momentum within the discretized domain, enabling the observation of secondary flow patterns and recirculation zones. This computational approach provides a high-fidelity representation of the hemodynamic environment, enabling the identification of regions where wall shear stress varies.

The Navier–Stokes equations describe the conservation of momentum and mass in the flow being studied. This allows us to predict the velocity and pressures that may be generated. Equation (1) describes the conservation of mass, while Equation (2) describes the conservation of momentum:

$$\nabla \cdot u = 0 \quad (1)$$

$$\rho \left(\frac{\partial u}{\partial t} + u \cdot \nabla u \right) = -\nabla p + \mu \nabla^2 u + f \quad (2)$$

where u is the fluid velocity vector; p is the pressure; ρ is the density of the blood; μ is the viscosity of the blood; and f is the body force, which in this case is considered to be zero. The equation stipulates that the mass entering a differential volume must equal the mass

exiting it, reflecting the principle of conservation of mass for an incompressible fluid. Its differential form is defined in Equation (3):

$$\frac{\partial u}{\partial x} + \frac{\partial v}{\partial y} + \frac{\partial w}{\partial z} = 0 \quad (3)$$

The expression represents a system of coupled, nonlinear partial differential equations that govern fluid flow motion across all spatial dimensions [19]. The directional components help to define the conservation of momentum within the arterial domain. These are defined in Equations (4)–(6).

X-axis:

$$\rho \left(\frac{\partial u}{\partial t} + u \frac{\partial u}{\partial x} + v \frac{\partial u}{\partial y} + w \frac{\partial u}{\partial z} \right) = -\frac{\partial p}{\partial x} + \rho g_x + \mu \left(\frac{\partial^2 u}{\partial x^2} + \frac{\partial^2 u}{\partial y^2} + \frac{\partial^2 u}{\partial z^2} \right) \quad (4)$$

Y-axis:

$$\rho \left(\frac{\partial v}{\partial t} + u \frac{\partial v}{\partial x} + v \frac{\partial v}{\partial y} + w \frac{\partial v}{\partial z} \right) = -\frac{\partial p}{\partial y} + \rho g_y + \mu \left(\frac{\partial^2 v}{\partial x^2} + \frac{\partial^2 v}{\partial y^2} + \frac{\partial^2 v}{\partial z^2} \right) \quad (5)$$

Z-axis:

$$\rho \left(\frac{\partial w}{\partial t} + u \frac{\partial w}{\partial x} + v \frac{\partial w}{\partial y} + w \frac{\partial w}{\partial z} \right) = -\frac{\partial p}{\partial z} + \rho g_z + \mu \left(\frac{\partial^2 w}{\partial x^2} + \frac{\partial^2 w}{\partial y^2} + \frac{\partial^2 w}{\partial z^2} \right) \quad (6)$$

The terms on the left-hand side describe the fluid's acceleration. This behavior arises from the transient fluctuations inherent in pulsatile blood flow, which drive velocity changes across the arterial morphology. The right-hand side of the equations describes the driving forces of the flow. The force involved in this case is the natural hydrodynamic pressure gradient generated by blood flow. In this regard, viscosity arises from the fluid's resistance [19,20].

The resolution of these equations yields the distribution of the pressure and vector velocity vector fields. This allows the calculated fluid pressure to be exported as a mechanical load on the arterial surface for the structural stress analysis. The resultant pressure onto the arterial structure demonstrates the mechanical deformation of the arterial wall across the cardiac cycle [20]. Given the pulsatile nature of the inlet conditions, it is understood that the flow through the geometry is unsteady, oscillating between the systolic and the diastolic phases. Furthermore, integrating these hemodynamic loads into the uncoupled structural domain enables the identification of critical regions prone to mechanical failure or remodeling. By computing the Von Mises stress and displacement gradients across the arterial wall, the model quantifies how the internal pressure pulses translate into cyclic strain [21].

Consequently, this computational framework provides a high-fidelity approximation of physiological hemodynamics. The numerical solution predicts flow disturbances caused by the bifurcation, where the branching geometry alters fluid stability. The separation of the flow leads to recirculation zones where velocity decreases. This commonly occurs in the carotid sinus region. These zones are characterized by localized variations in static pressure and velocity magnitudes. These effects are critical indicators that alter the WSS distribution, highlighting potential zones for plaque accumulation [21].

Once the geometries for analysis are available, the discretization must be generated. Given that the bifurcation area experiences severe gradients in velocity, pressure, and WSS, the mesh prioritized a higher density of nodes and elements in this critical region (Figure 6) [22]. It is considered that the discretization of the control volume should have

a larger number at the bifurcation, so in this case, a sphere of influence will be applied. This operation allows a sphere of a given radius to be placed, with the nodes originating in a specific area. A coordinate system was positioned at the center of the bifurcation to concentrate the mesh density at this area. Implementing the sphere in the bifurcation region created a refinement that concentrates the most nodes. It was decided to perform a local refinement given the available computational capacity; therefore, performing a uniform refinement throughout the control volume would require significantly more computational resources. Additionally, inflation layers were specified along the fluid boundaries to achieve greater precision in flow behavior. This configuration enabled the solver to precisely resolve the viscous shear effects driving the WSS gradients. Specifically, fifteen inflation layers were applied with a height of 0.8 mm and a growth rate of 1.31. This generates a total of 180,280 nodes and 497,123 elements for the discretization.

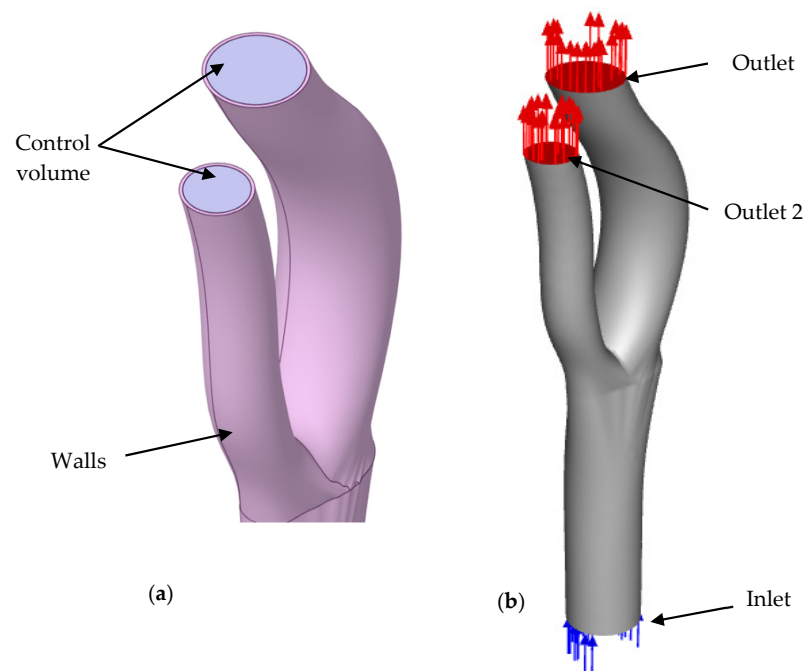


Figure 5. Artery structure. (a) Artery walls and volume control. (b) Boundary conditions for the volume control. The fluid inlet is represented by blue arrows. Red arrows indicate the fluid outlets.

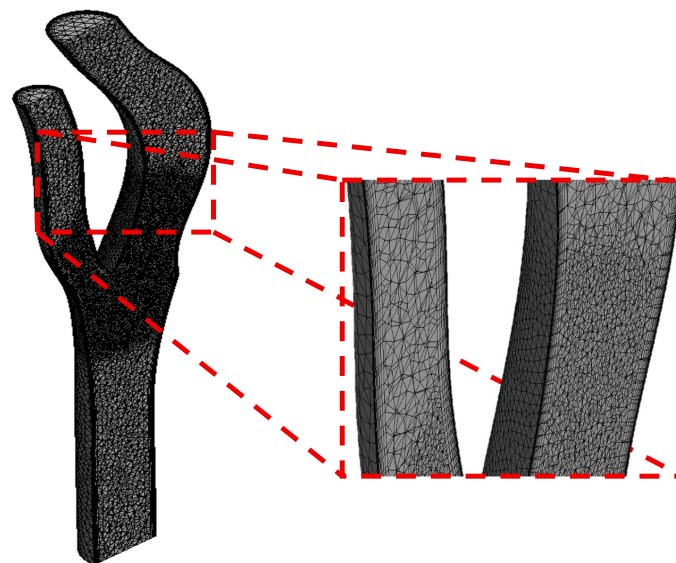


Figure 6. Volume control meshing and close up.

To ensure that the numerical solutions were independent of the spatial discretization, a mesh-independence study was conducted. This process guarantees that the resultant velocity and pressure fields reflect a precise behavior rather than numerical artifacts. The spatial domain was discretized using progressively refined grids, and mesh convergence was evaluated against specific quality metrics. As presented in Table 1, the mesh characteristics are evaluated by tracking skewness as a function of both the global element size and the localized refinement via a sphere of influence.

Table 1. Mesh independence study for control volume.

Mesh	Element Size (mm)	Sphere of Influence Element Size (mm)	Element Quantity	Node Quantity	Max Velocity m/s	Skewness
A	3	2	22,187	10,481	1.343	0.54–0.63
B	2	1	71,316	31,139	1.463	0.14–0.23
C	1.5	0.5	270,178	102,540	1.476	0.13–0.22
D	0.9	0.2	497,123	180,280	1.487	0.05–0.14

For the subsequent structural analysis, the computed pressure fields served as the mechanical loading conditions applied directly onto the internal arterial wall. The discretization was performed exclusively for the solid arterial domain. The structural mesh isolates the wall and excludes the internal volume. A total of 212,439 nodes and 105,318 elements were generated in the discretization of the geometry (Figure 7). This mesh density was selected to ensure numerical convergence while capturing the stress gradient across the vessel's curvature. Maintaining this nodal resolution enabled the structural solver to precisely identify critical mechanical demand points at the carotid bifurcation. Consequently, the simulation provides a robust framework for assessing the structural integrity of the arterial walls under peak loading conditions. This discretization ensures that structural response governed by the material properties and resultant pressure is accurately represented throughout the cardiac cycle.

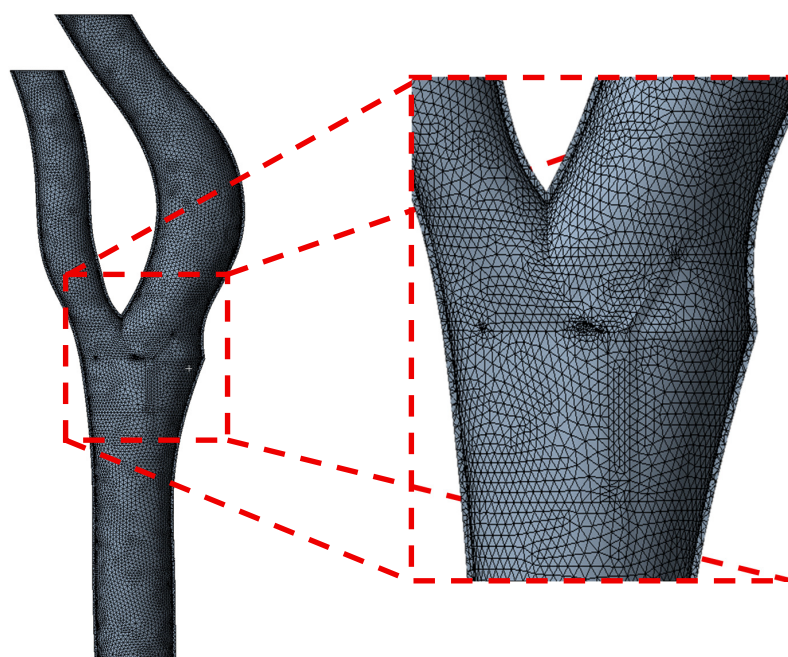


Figure 7. Arterial wall meshing for structural analysis.

A mesh independence study was conducted for the structural analysis by tracking the maximum Von Mises stress as the primary convergence variable. As detailed in Table 2,

the solid domain was discretized using progressively refined grids defined by the global element size and localized volumetric refinement. Furthermore, mesh conditioning was strictly monitored via the orthogonal quality metric, ensuring optimal geometric limits to minimize discretization errors and secure robust solver convergence.

Table 2. Mesh independence study for structural geometry.

Mesh	Element Size (mm)	Element Quantity	Node Quantity	Max Von Mises Stress (MPa)	Orthogonal Quality
A	3	15,569	31,503	0.134	0.44–0.54
B	2	28,811	57,849	0.157	0.53–0.63
C	1.5	47,690	94,938	0.189	0.64–0.74
D	0.9	105,318	212,439	0.237	0.74–0.84

3.2. Boundary Conditions for Fluid Analysis

In computational hemodynamics, blood must be accurately characterized to reflect its complex physiological behavior. In this study, blood is modeled as a non-Newtonian, incompressible fluid, a distinction crucial for capturing the shear-thinning properties where viscosity decreases under higher shear rates, a common occurrence in the carotid bifurcation. This paper was based on Carreau’s model to accurately simulate blood flow behavior through the artery [23]. Furthermore, the flow is characterized by a low Reynolds number (Re) presented in Equation (7), which justifies the assumption of a laminar flow regime, as inertial forces are dominated by viscous forces within the arterial lumen. Blood flow in the carotid artery typically remains at a low Reynolds number, reaching a peak of 1000. For this study, a value of 200 was used [24,25].

$$Re = \frac{\rho U D_h}{\mu} \quad (7)$$

where ρ is the density of the blood, U is the fluid velocity, D_h is the hydraulic diameter, and μ is the viscosity of the blood.

The physical properties utilized for the simulation, including the dynamic viscosity and constant density, are detailed in Table 3. These parameters were evaluated at two critical phases of the cardiac cycle: the systolic peak, corresponding to maximum flow velocity and pressure, and the diastolic peak, which corresponds to the minimum flow velocity and pressure. This dual-point sequential analysis ensures a comprehensive assessment of the hemodynamic environment and its mechanical impact on the uncoupled arterial wall throughout the pulse wave.

Table 3. Mechanical properties of blood.

Concept	Variable		References
	Systolic Peak	Diastolic Peak	
Blood density	1050 kg/m ³	1050 kg/m ³	[26]
Blood velocity	1.15 m/s	0.32 m/s	[27]
Dynamic viscosity of blood	0.0055 Pa-s	0.0023 Pa-s	[28]
Kinematic viscosity of blood	3.8×10^{-6} m ² /s	3.8×10^{-6} m ² /s	[29]

A flat uniform velocity profile was selected at the CCA inlet; notably, the computational control volume lacks an upstream inlet extension length, meaning flow development occurs downstream within the reconstructed geometry. The fluid density will remain constant throughout the path, mainly due to the conservation of mass present in the flow. The arterial walls were assumed to be rigid and with no slip condition. The physiological

cardiac cycle spans a period of 0.8 s; this framework models the peak systolic and diastolic states. Anatomically, the internal carotid artery (ICA) carries the greater volume of blood to the brain; therefore, the blood flow distribution between the ICA and the external carotid artery (ECA) is estimated to be 70:30.

3.3. Boundary Conditions for Structural Analysis

Blood flow alters the structure of the artery through the pressures it generates. The forces acting internally lack a direction and are reflected from different directions onto the arterial walls (Figure 8) [30]. The pressure deforms the artery, briefly altering its structure. However, arteries generally behave viscoelastically, reaching large deformations, but due to their composition, they can return to their original state. Considering the artery's position, it is understood that its movement is limited along most of its length. However, the vessel undergoes localized radial expansion and contraction during the pressure pulse, justifying the application of fixed boundary conditions [31]. To evaluate the structural behavior, one-way coupling was implemented to develop the structural analysis according to the CFD pressure distribution into the structural solver.

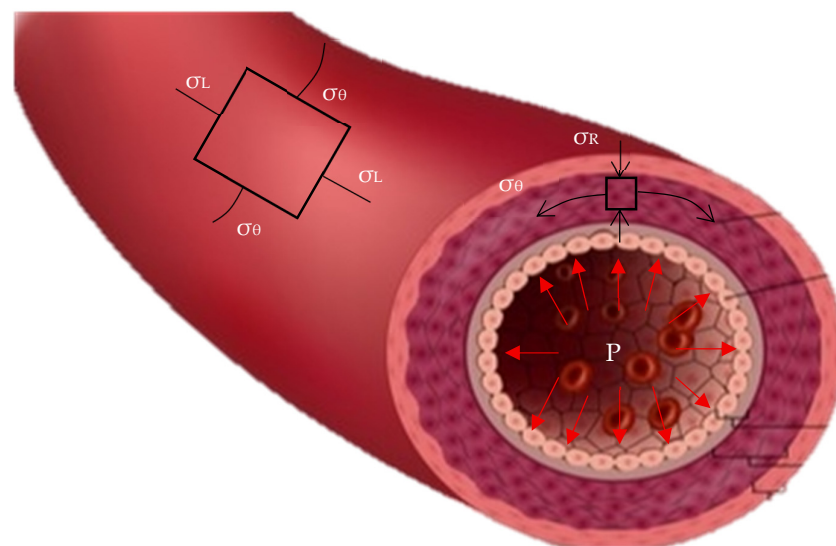


Figure 8. Load configuration in the artery. Red arrows represent the distribution of blood pressure within the artery. Black squares show the orientation and distribution of radial and longitudinal stresses throughout the vessel's structure.

To approximate the physiological behavior of the vessel, the mechanical properties of the structural domain were modeled, considering only two walls, the intima and the media, exposing them as a single wall. Tables 4 and 5 outline the material parameters utilized for the numerical simulation. This structural framework considered the artery's structure as if it behaved linearly with viscoelastic additives. The integration of these constitutive properties is fundamental to capturing the effects during the cardiac cycle, ensuring that the structural domain accurately resolves the strain distribution under the mapped peak systolic and diastolic pressure fields.

Table 4. Isotropic properties for artery walls during systolic and diastolic peak.

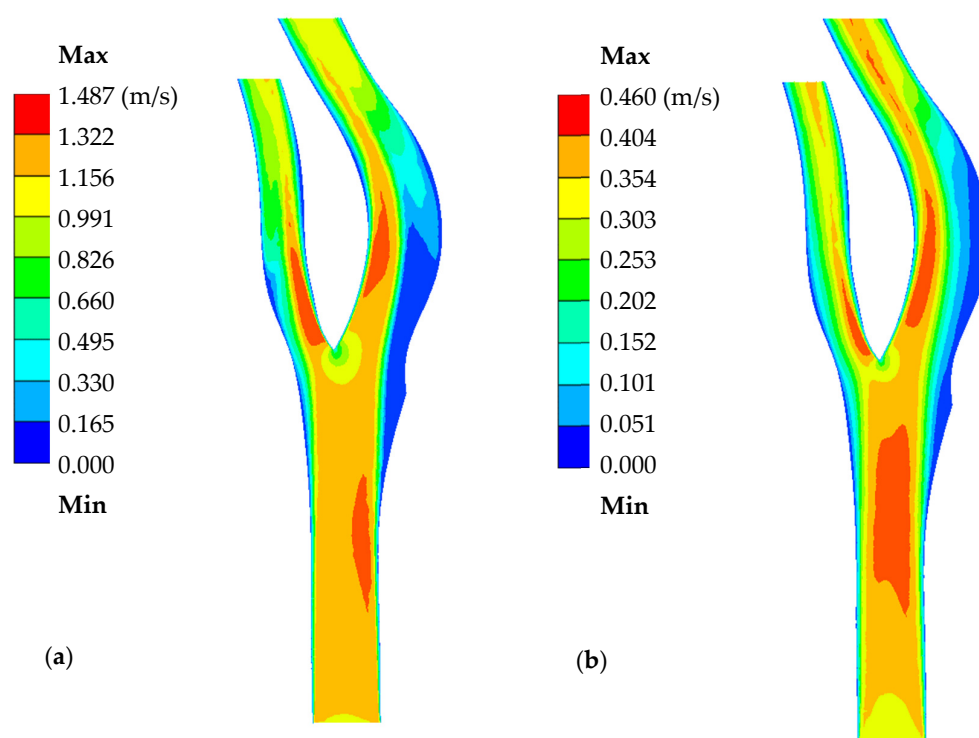
Concept	Variable	Reference
Young's Modulus (MPa)	2.05	[32]
Poisson ratio	0.49	[33]
Wall density (kg/m ³)	1120	[26]

Table 5. Viscoelastic properties for artery walls during systolic and diastolic peak.

	$i = 1$	$i = 2$	$i = 3$	$i = 4$	$i = 5$	Reference
Relative moduli	6.28×10^{-13}	0.53	7.15×10^{-15}	0.47	3.67×10^{-16}	[34]
Relaxation time (s)	10.90	60.85	585.55	627.35	886.51	

3.4. CFD Analysis Results

Figure 9 illustrates the blood velocity profiles within the carotid artery during the systolic (a) and diastolic (b) peaks. Based on the inlet boundary conditions specified in Table 3, the velocity developed along the CCA length. Notably, localized velocity reductions and flow deceleration occur primarily within the carotid sinus and the proximal segments of the ICA and ECA.

**Figure 9.** Velocity contour of the artery. (a) Systolic peak. (b) Diastolic peak.

This deceleration is directly attributed to the complex branching geometry, which directs the flow predominantly toward the flow divider of the bifurcation [35]. A comparative analysis reveals that the flow behavior fluctuates between the systolic and diastolic phases; specifically, the regions of low velocity and recirculation are less extensive during the diastolic peak than during the systolic peak. This is consistent with the behavior described by Ku, in which the bifurcation geometry causes decreases in flow velocity [36]. Quantitatively, the maximum recorded velocities were 1.487 m/s and 0.460 m/s for the systolic and diastolic peaks, respectively.

Figure 10 illustrates the distribution of static blood pressure within the arterial fluid model. In accordance with fluid dynamics principles, pressure and flow velocity are inversely related; areas of higher velocity correspond to lower pressure gradients, and vice versa. Significant pressure fluctuations are observed along the arterial extension, particularly at the bifurcation, as described by Magder et al. [36]. At the bifurcation, the sudden changes in cross-sectional area and the presence of the carotid sinus induce localized pressure gradients [37]. During the systolic peak, these pressure variations are

most pronounced at the origin of the ECA and along the medial section of the CCA, coinciding with regions of maximum velocity.

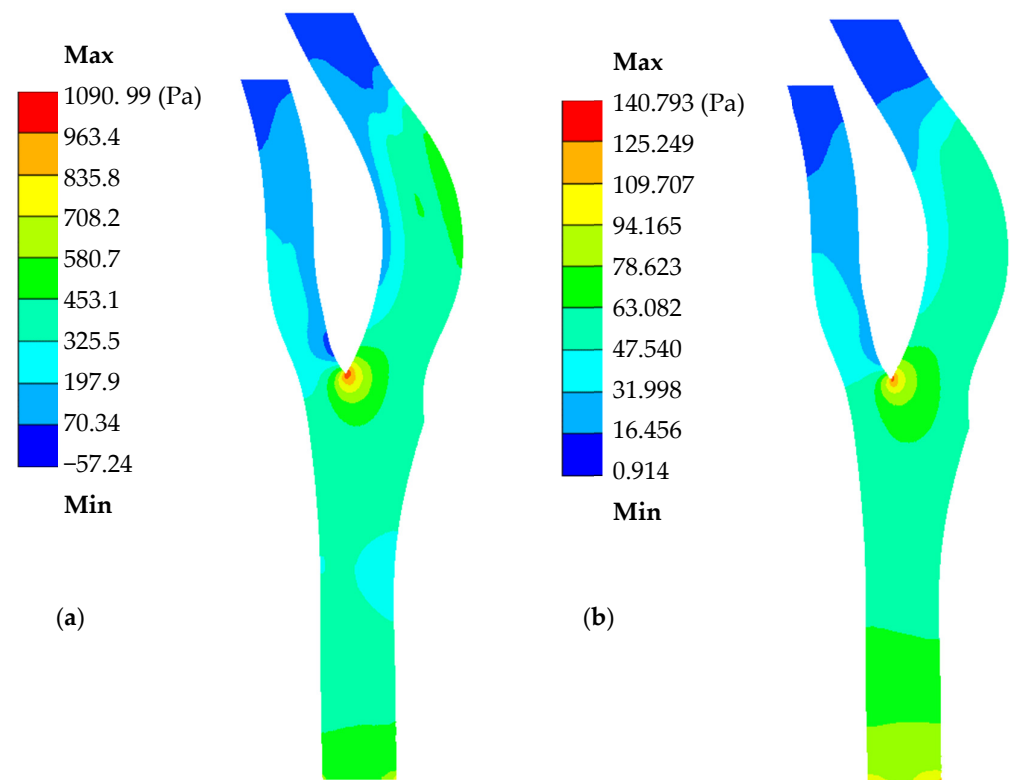


Figure 10. Pressure contour of the artery. (a) Systolic peak. (b) Diastolic peak.

The diastolic peak displays a comparable distribution pattern, although with localized changes at the proximal ICA, ECA, and the mid-section of the CCA. Quantitatively, the systolic phase reaches a maximum pressure of 1090.99 Pa, whereas the diastolic phase recorded a maximum of 140.793 Pa.

A direct correlation exists between flow velocity and the generation of wall shear stress (WSS), resulting from the frictional forces exerted by the fluid along the rigid arterial boundaries. In vascular hemodynamics, regions characterized by lower velocities or disturbed flow patterns are often associated with an increased probability of lipid accumulation and atherosclerotic plaque formation, as Rezazadeh notes in his research on the relationship between atherogenesis and low WSS [35,38]. While this is not a direct indicator of atherosclerosis, it does contribute to our understanding of the areas where it can develop. Figure 11 illustrates the spatial WSS distribution across the arterial domain, derived from the previously computed velocity profiles.

The WSS distribution primarily reflects regions characterized by significant hemodynamic fluctuations and velocity gradients. In both analyzed phases, WSS along the CCA remains low and uniform, a behavior attributed to the stable laminar flow and steady velocity [39]. During the systolic peak, the ICA exhibits a progressive increase in WSS along its length; however, a marked reduction is observed within the carotid sinus. This localized decrease reflects the flow separation and recirculation zones induced by the abrupt geometric expansion of the sinus. A similar trend is observed in the ECA, where low WSS regions correlate with areas of reduced velocity, consistent with the findings of Katritsis et al. [40]. The bifurcation represents the most critical region, experiencing the highest gradients in stress magnitude. Although the diastolic peak exhibits a comparable spatial distribution pattern, the overall magnitudes and the extent of these low-stress zones

are diminished. Notably, the maximum WSS values recorded at the immediate CCA inlet in both scenarios are identified as numerical artifacts. This is mainly due to the lack of a control volume extension at the inlet, the sudden transition from a flat inlet profile to the wall no-slip boundary condition generates an unphysiological, localized spike before the flow can adjust downstream.

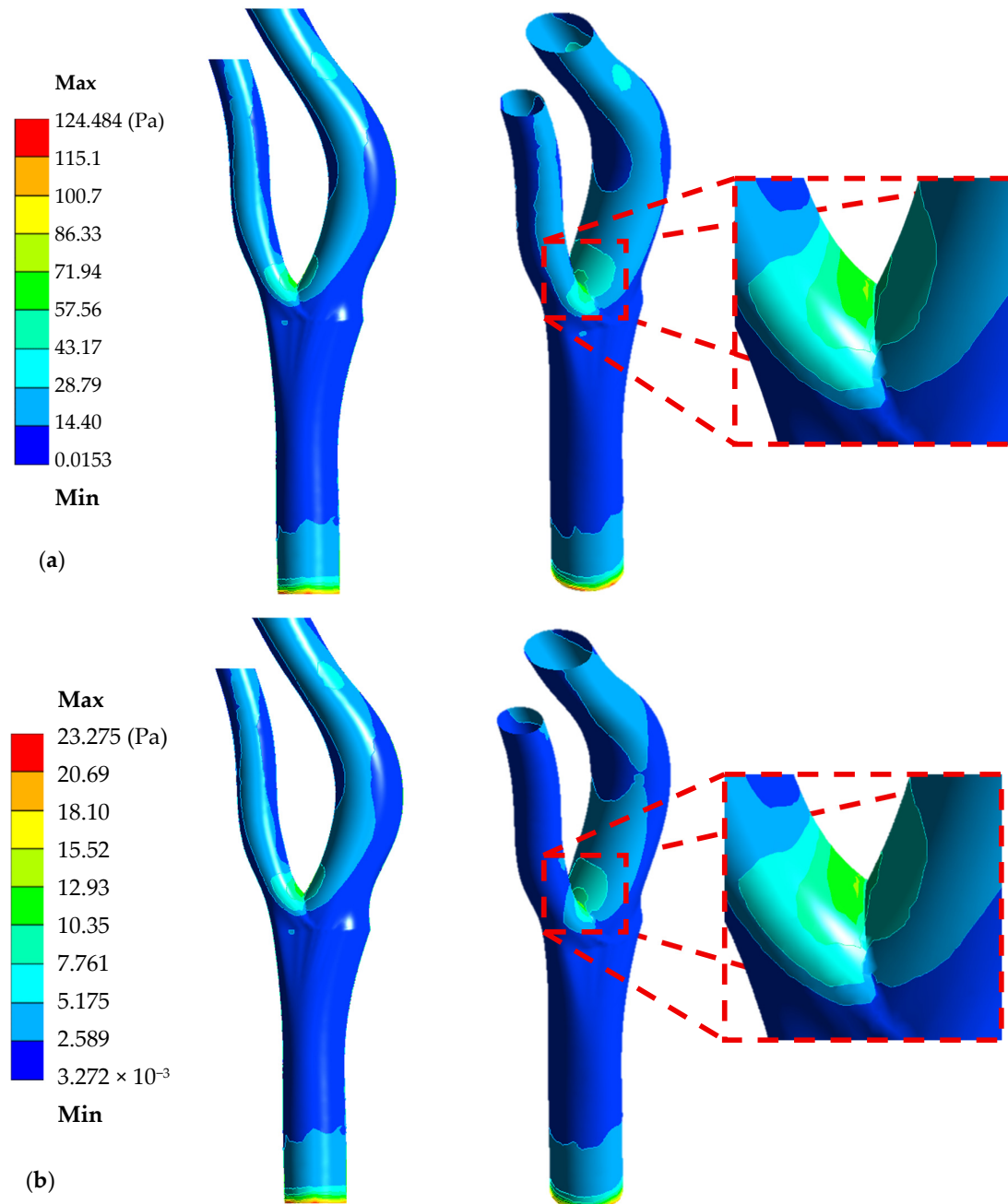


Figure 11. Wall shear stress contour of the artery. (a) Systolic peak. (b) Diastolic peak.

3.5. Structural Analysis Results

For the structural analysis, tests were performed according to the peaks presented (systolic and diastolic). The results for total displacement and Von Mises stress are presented in different views and approaches to understand how the structure is affected. It should be noted that the properties used for the analysis considered linear viscoelastic behavior.

Figure 12 illustrates the total displacement experienced by the arterial structure during the systolic peak. As shown, the maximum displacement magnitudes are localized in

the bifurcation zone and along the ICA and ECA branches. As this region undergoes severe hemodynamic transitions (such as increased pressure and decreased velocity), it is understandable that the displacement is high. According to Gepner et al. description, the carotid artery can undergo significant displacement, up to 0.5 cm, depending on its wall structure tethering [41]. Furthermore, Zhu et al. demonstrated through clinical analysis that the carotid artery exhibits distinct longitudinal translations during cardiac cycle [42]. Given the model's properties, including viscoelasticity and based on clinical cases, it is possible that arteries attain these dimensions during normal displacement in a healthy state [43].

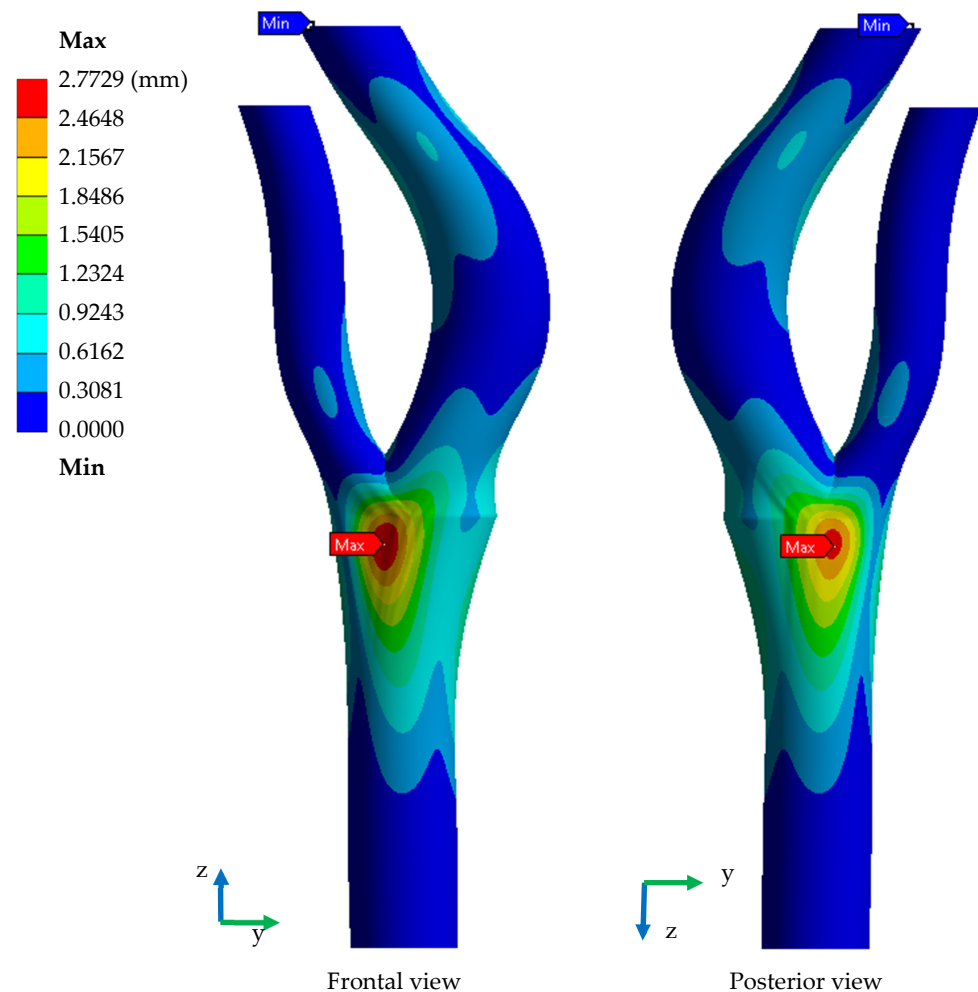


Figure 12. Total displacement during systolic peak. The z and y axes show the position of the plane in which the result was captured, in order to highlight the most relevant areas.

Figure 13 illustrates the displacement behavior during the diastolic peak. The magnitude decreases significantly, representing the passive elastic recoil of the artery as both internal pressure and fluid velocity decrease. At this stage, the vessel returns to its baseline geometric state for a brief period before the next pulse.

Figure 14 illustrates the distribution of Von Mises stress during the systolic peak in the arterial wall. The stress contours are predominantly concentrated at the carotid bifurcation, with a maximum value of 0.2375 MPa. This magnitude remains below the ultimate tensile strength of arterial tissue, indicating structural integrity under simulated physiological loads, as Teng et al. reported in their experimental investigation [44]. This structural response is evident in both the external and internal laminae; the stress gradients are localized on the internal luminal surfaces. The peak resultant stress occurs internally, characterized

by a stress concentration along specific internal geometries. While ICA presents minor secondary stress increments along its longitudinal axis, these do not approach the intensity observed at the bifurcation, which remains the primary site of mechanical demand.

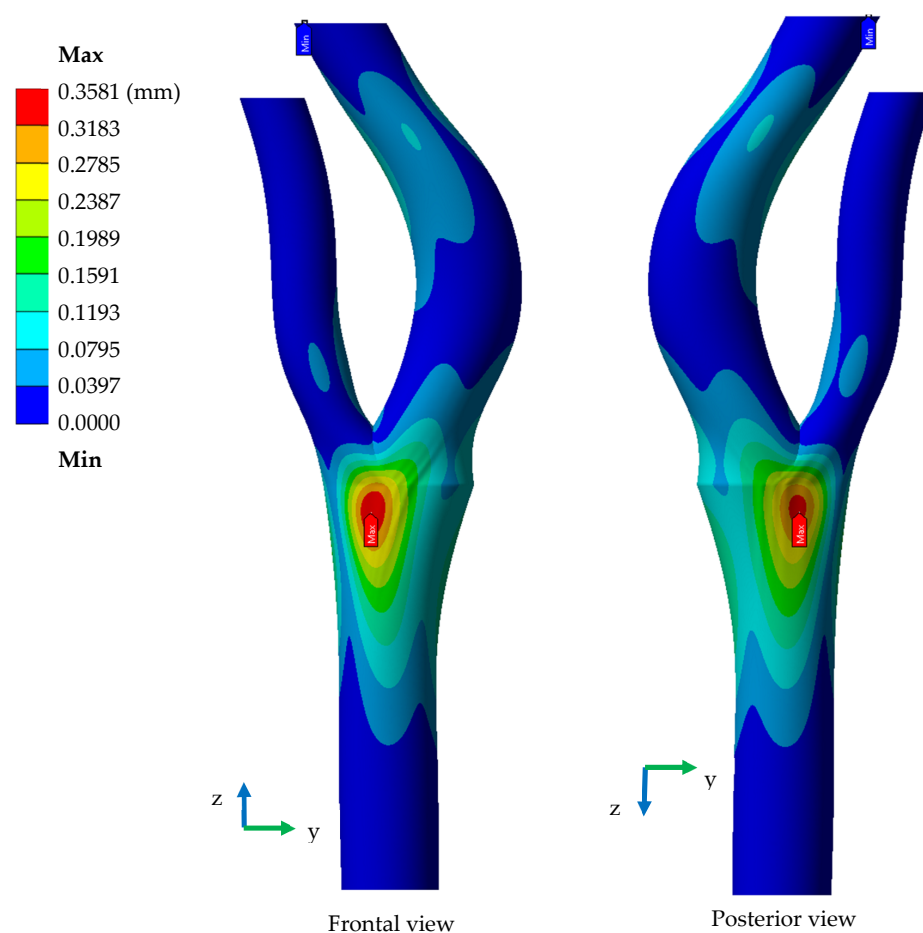


Figure 13. Total displacement during diastolic peak. The z and y axes show the position of the plane in which the artery is located, in order to capture the effects produced on its frontal and posterior surfaces.

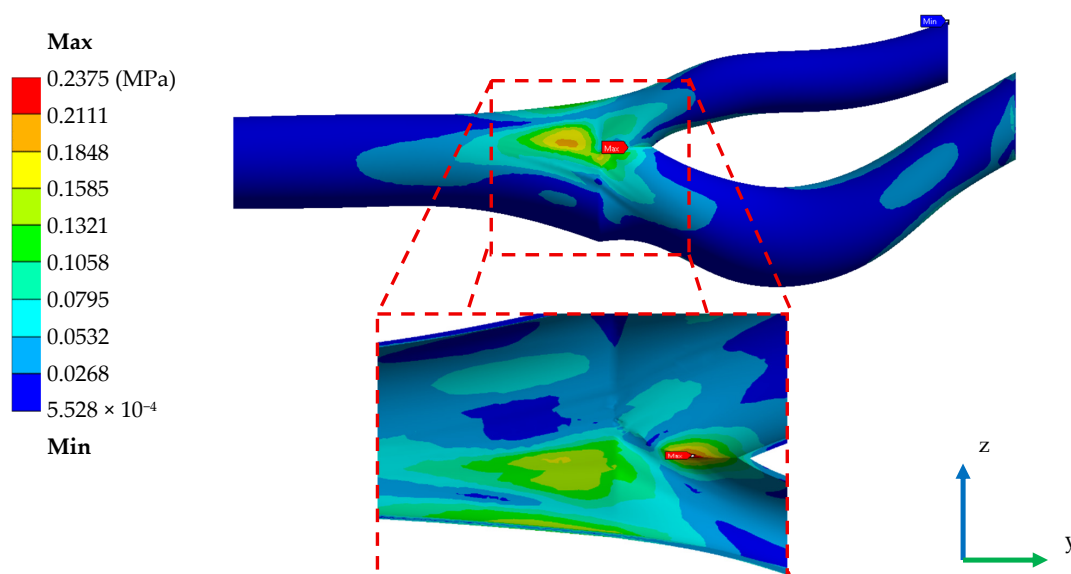


Figure 14. Von Mises stress during systolic peak and close up to its internal wall. The plane where the results are captured is highlighted by the z and y axes, in order to identify the areas of interest.

Figure 15 illustrates the structural response and Von Mises stress distribution of the arterial wall during the diastolic peak. The overall mechanical behavior remains consistent with the patterns observed during the systolic peak; however, it is characterized by significantly lower magnitudes. It reaches a maximum magnitude of 0.0306 MPa, localized at the point of geometric variation, the carotid bifurcation.

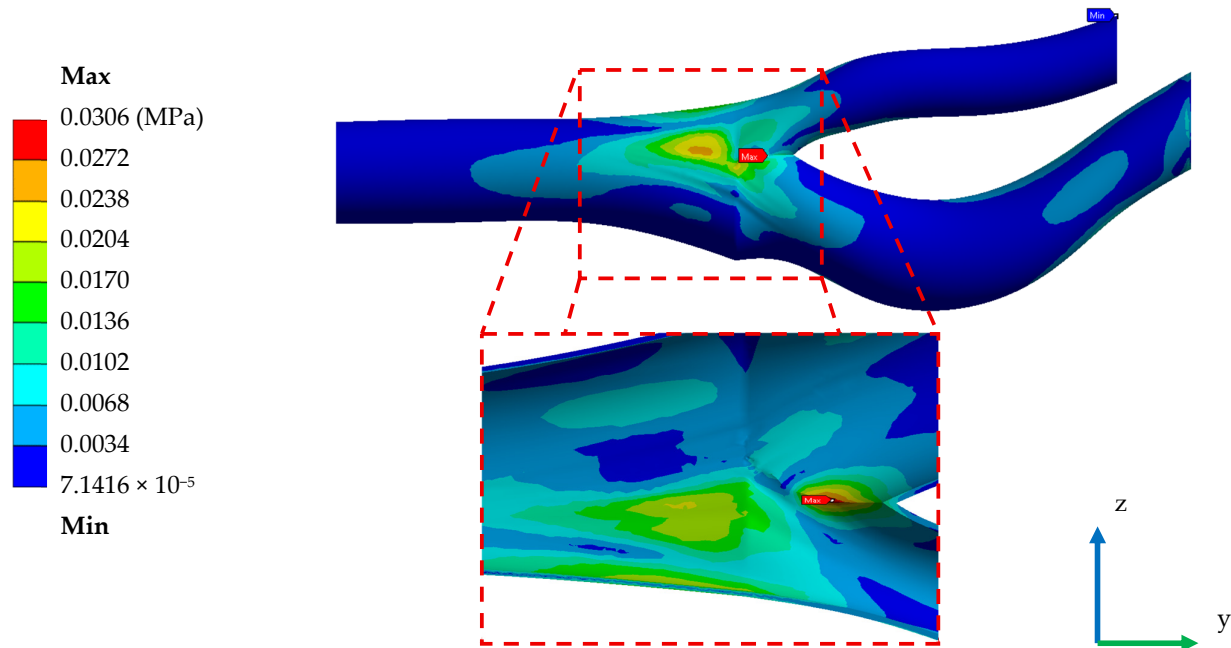


Figure 15. Von Mises stress during diastolic peak and close up to its internal wall. The z and y axes highlight the plane emphasizing the areas of interest.

The carotid bifurcation represents the most critical zone of the geometry, where both hemodynamic and structural concentrations are present. Due to sudden branching, this region is subject to complex mechanical loading. The arterial wall undergoes cyclical variations between the systolic and diastolic peaks, influencing its behavior in response to blood flow, a phenomenon that aligns with the fundamental studies by Liepsh et al. and Motomiya et al. [45,46]. Furthermore, alterations in the intima-media thickness can alter the extension of the carotid artery, thereby modulating its passive structural response and deformation gradients under physiological pressure profiles.

4. Discussion

The analysis of blood behavior and its effects on arterial wall structures has been a growing area of research in recent years. In light of this, different methodologies and results have been presented on how these analyses are performed. This research was based on various studies that demonstrate how blood flows through the arteries. Some studies typically reconstruct the geometry of the carotid artery based on dimensions derived from clinical analyses. In contrast, this study used MRI images to reconstruct the carotid artery geometry with greater precision. The work carried out demonstrates progress in conducting this type of study. The results obtained are similar, with the distinction that the geometry was generated from images, unlike the work by Bashar et al. and Shahzad et al., and that the properties of the walls were taken into account for a structural analysis [47,48]. For this study, one objective is to demonstrate the effects on the artery when it is involved in the changes generated by the bifurcation. As shown in the Results section, both the fluid and structural results indicate that the change generated at the bifurcation produces a series of effects on the structure that can affect it in different ways. In this study, a flat

uniform velocity profile was selected at the CCA inlet without an extension length. This approach represents a limitation, as it is known to generate values and gradient artifacts in the immediate vicinity of the inlet boundary. However, this localized numerical effect did not compromise the overall hemodynamics assessment. The fluid flow behavior and the resulting hemodynamic pattern near the carotid bifurcation, such as flow separation zones and WSS distributions, realigned and demonstrated strong qualitative and quantitative agreement with trends reported in similar numerical and experimental studies as described in the numerical study conducted by Rezazadeh et al. and the experimental and clinical analyses by Paszkowiak et al. and Katritsis et al. [38–40]. In terms of flow, an effect can occur in the arteries that alters structure when fluid behavior changes, as well as the effect generated by wall shear stress. Figure 9 shows how, in the ICA and to a lesser extent in the ECA, there is a decrease in velocity due to the change in geometry generated by the bifurcation. However, this effect generates turbulence and reduces wall shear stress. One point to consider is that turbulence in the artery and low WSS may contribute to, or partially indicate, regions where lipid accumulation could occur. In terms of arterial structure, pressure-induced stresses primarily affect the bifurcation region. As described in the study by Spanos et al., the forces acting on the arterial walls are significant because they can contribute to the development of atherosclerosis [49]. Based on the observed results, the artery appears to exhibit these effects in certain regions, such as the ICA and ECA sinuses. This is similar to the study by Chao et al., which describes how blood flow velocity alters the distribution of WSS in the carotid artery [50]. However, the latter study adds an obstruction in the artery. One of the important points of this research is the consideration of the structural effects in arterial walls, in addition to their mechanical properties. There are studies that examine the effects of arterial structure. However, the analysis conditions are rarely considered, as the properties are often treated as linear hyperelastic, with hyperelasticity ignored and linearity reflected.

5. Limitations

A significant limitation of the present study lies in the simplified, uncoupled, one-way pressure-to-structure mapping. In Section 3.2, the fluid domain was solved assuming strictly rigid arterial walls, whereas Section 3.5 resolves structural displacements reaching a peak magnitude of 2.77 mm during systole. From a strict continuum mechanics perspective, solving the hemodynamic fields over a non-deformable boundary neglects the compliance of the vessel wall. The biomechanical literature demonstrates that treating the fluid domain as rigid overestimates peak systolic pressure gradients and wall shear stress (WSS) magnitudes by approximately 15% to 25% compared to fully coupled multi-directional Fluid–Structure Interaction (FSI) models using Arbitrary Lagrangian–Eulerian (ALE) formulations, as Torii et al. and Formaggia et al. mentioned in their studies about fluid dynamics in different vessels [51,52]. This overestimation occurs because a rigid wall lacks the capacity to absorb kinetic energy via radial expansion, thereby driving higher hydrostatic loads onto the subsequent structural solver. Consequently, the absolute displacement peak of 2.77 mm reported herein must be interpreted as a conservative upper-bound estimation—a ‘worst-case scenario’ mechanical evaluation. Furthermore, these results are consistent with established clinical benchmarks and experimental evidence from different studies [41,42]. While the rigid fluid boundary condition introduces a systematic overestimation in the absolute stress and strain magnitudes, extensive comparative biomechanical studies confirm that one-way uncoupled frameworks preserve the spatial topology, contour distributions, and geometric localization of critical mechanical demand points (such as stress concentrations at the carotid bifurcation) with high fidelity. Therefore, while the absolute quantitative values are amplified by the systematic error inherent to the

rigid fluid assumption, the structural conclusions regarding the site of primary mechanical vulnerability and the structural integrity margin of the tissue remain qualitatively robust and clinically relevant for comparative risk assessments.

6. Conclusions

Advances in hemodynamics have enabled the analysis of different arteries, providing a greater understanding of their behavior. This study focuses on the carotid artery and presents results on blood flow and arterial walls. The results shown allow us to understand how blood flow is affected by changes in geometry, such as a bifurcation. A series of changes affects the behavior of the blood, and the structure of the artery. The analyses performed at the systolic and diastolic peaks reveal the drastic difference between the two. The systolic peak increases arterial displacement, with effects mainly concentrated at the bifurcation. The effect on the artery can manifest in various ways. The biological tissues that make up arteries can be severely affected by the high stresses caused by blood pressure. However, the material's regenerative properties allow it to remodel, causing the walls to rebuild themselves. This same effect alters the material's mechanical properties, making them more resistant to the stresses caused by blood flow. The results accurately depict flow behavior consistent with what is expected from a healthy model without obstruction or changes in blood composition. Furthermore, the results are consistent with those reported in other studies [47,51]. The structural analysis highlights key points, demonstrating that concentration occurs primarily at the bifurcation, a point of interest for future research. However, this study has some limitations. One of these is the coupling between the fluid and the structural component, as it does not account for movement in the arterial structure; therefore, this can be considered a shortcoming in terms of the desired level of accuracy. Additionally, this analysis has limitations in the structural component, as the use of an elastic model with viscoelastic behavior does not achieve the highest possible accuracy in approximating the artery's actual behavior. Since the actual artery behaves as an anisotropic material, the model could be modified in future research to provide a more precise structural analysis.

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Abbreviations

The following abbreviations are used in this manuscript:

CAT	Computed Axial Tomography
MRI	Magnetic Resonance Imaging
CCA	Common Carotid Artery
ICA	Internal Carotid Artery
ECA	External Carotid Artery
CFD	Computational Fluid Dynamics
FEM	Finite Element Method
CAD	Computer-Aided Design
WSS	Wall Shear Stress

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