

**INTERNATIONAL CONFERENCE AND ADVANCED WORKSHOP  
ON MODELING AND SIMULATION OF COMPLEX SYSTEMS**

**Biomechanical Analysis of Hemodynamic  
Muscular Pressure on the Arterial Blood  
Vessel: Insights from Computational Modeling**

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**Research paper Presentation**

**By**

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# Presentation outline

- \* Abstract:
- \* Introduction
- \* Justification of the study
- \* Methodology
- \* Results, discussion of findings, contribution, recommendation
- \* Conclusion

## **Abstract:**

**High blood pressure, also known as hypertension, is a common medical condition where the force of blood against the walls of the arteries is consistently too high. During the last 100 years, pathological methods have been used to explain different ways that recapitulate the unique features of muscles and how they relate to the arterial blood vessel. The mechanical characterization of pressure acting on the artery blood vessel when the muscle collapses has resulted in high blood pressure. The biomechanical environment causes behavioral changes that affect the wall of the vessel. This is due to the mechanical properties applied to the artery to obtain stress relaxation, burst pressure on the valves, and dynamics biomechanical analysis. These resulted in the non-linearity and hysteresis of the blood flow. A consistent biomechanical contract of the muscles on the artery blood vessel can lead to high blood pressure, which can lead to death. This can be subdued by in-depth analysis using a computational technique to investigate the missing element that has resulted in physiological and biomechanical performance on the arterial blood vessel by the contracted muscles. The result shows the hemodynamic factor to be  $6.6e^{-7}$  that help reduce high blood pressure. These models will help researchers and clinicians understand the complex hemodynamics and mechanics of blood flow, predicting the effects of various physiological and pathological conditions, and developing new diagnostic and treatment strategies for cardiovascular diseases.**

**Keywords: High blood pressure; Biomechanical model; Static structural analysis; Finite element, Hemodynamics factor ;**

# Introduction

High blood pressure, also known as hypertension, is a common medical condition where the force of blood against the walls of the arteries is consistently too high [1].

Blood pressure is measured using two values: systolic pressure (the top number) and diastolic pressure (the bottom number). Normal blood pressure is typically around 120/80 millimeters of mercury (mmHg) [2]. Hypertension are of two types

**Primary hypertension:** This type of hypertension develops gradually over time and does not have a specific cause. It is the most common type and is often related to a combination of factors such as age, genetics, and lifestyle choices.

**Secondary hypertension:** This type of hypertension is caused by an underlying medical condition, such as kidney problems, hormonal disorders, or certain medications.

There are many risk factors such as age, family history and unhealthy lifestyle.

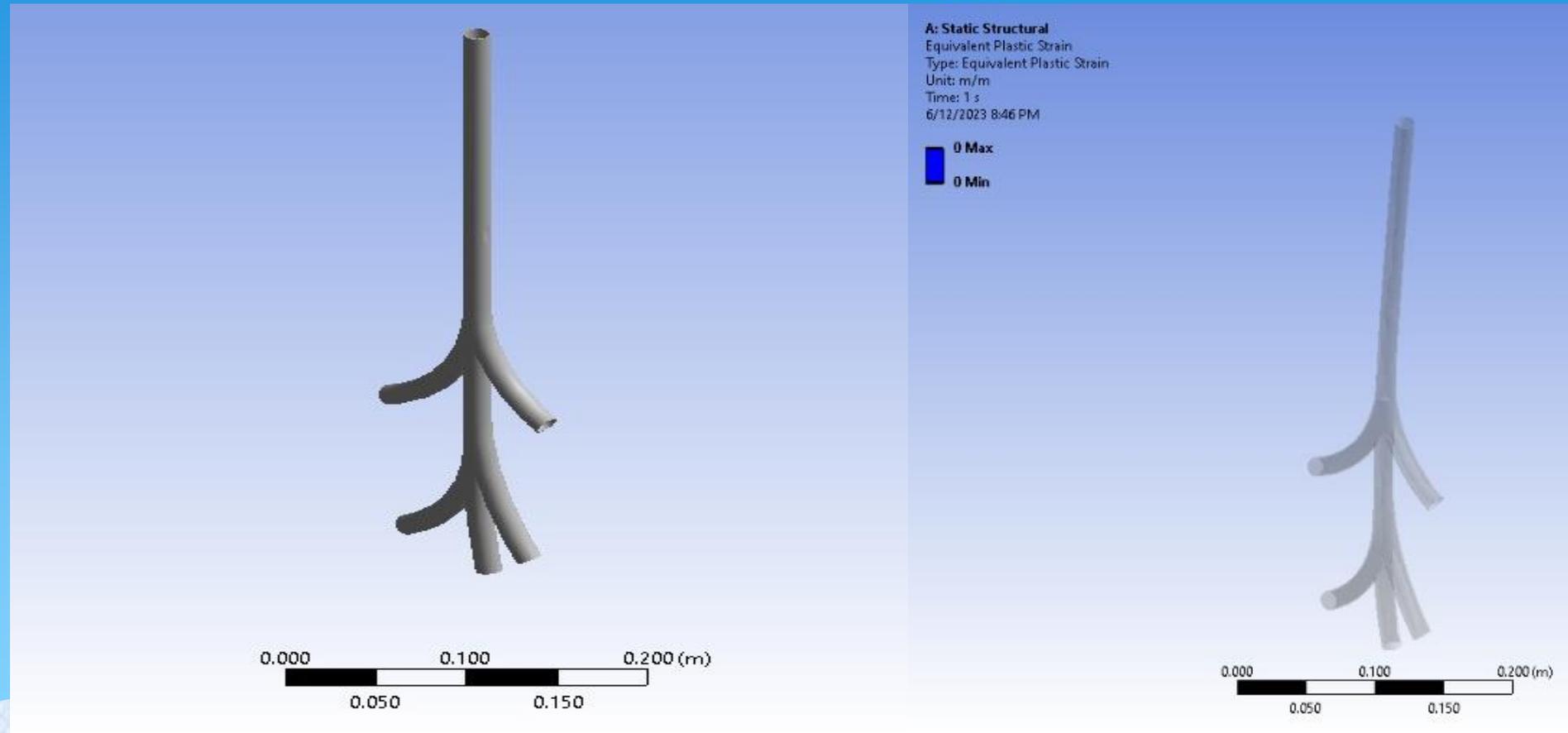
# Methodology

## CAD Modelling

A 3D model of the arterial vasculature was created on Solid works 2023 Software, typically based on anatomical data and simplified geometries of small arteries [27]. The analysis focuses on understanding how arteries respond to mechanical forces, such as blood pressure of 15mmHg and flow, and how their structural properties influence their function. There are several aspects of biomechanical analysis that are commonly investigated in artery blood vessels shown in Figure 1. The diameter of the artery blood vessels can vary among individuals and is influenced by various factors such as age. On average, the diameter of the artery blood vessel ranges from approximately 1 to 6 millimeters with wall thickness ( $\mu\text{m}$ ) between 40 to 500 and total length (mm) of 160000. Arteries are subjected to various mechanical forces, including blood pressure, pulsatile flow, and wall tension. Biomechanical analysis aims to quantify the mechanical properties of arteries, such as their stiffness, compliance, elasticity, and strength. This is often done by applying controlled loads to artery samples and measuring their resulting deformation or stress-strain relationships [24]. Biomechanical analysis was considered in the hemodynamic factors that influences artery function. This includes studying blood flow patterns, shear stresses, and pressure distributions within the arterial system. Computational fluid dynamics (CFD) simulations and flow visualization techniques was employed to investigate hemodynamic parameters.

# Methodology

## CAD Modelling



**Figure 1. 3D geometry of arterial vasculature (left) and Equivalent plastic strain (right)**

## **Methodology**

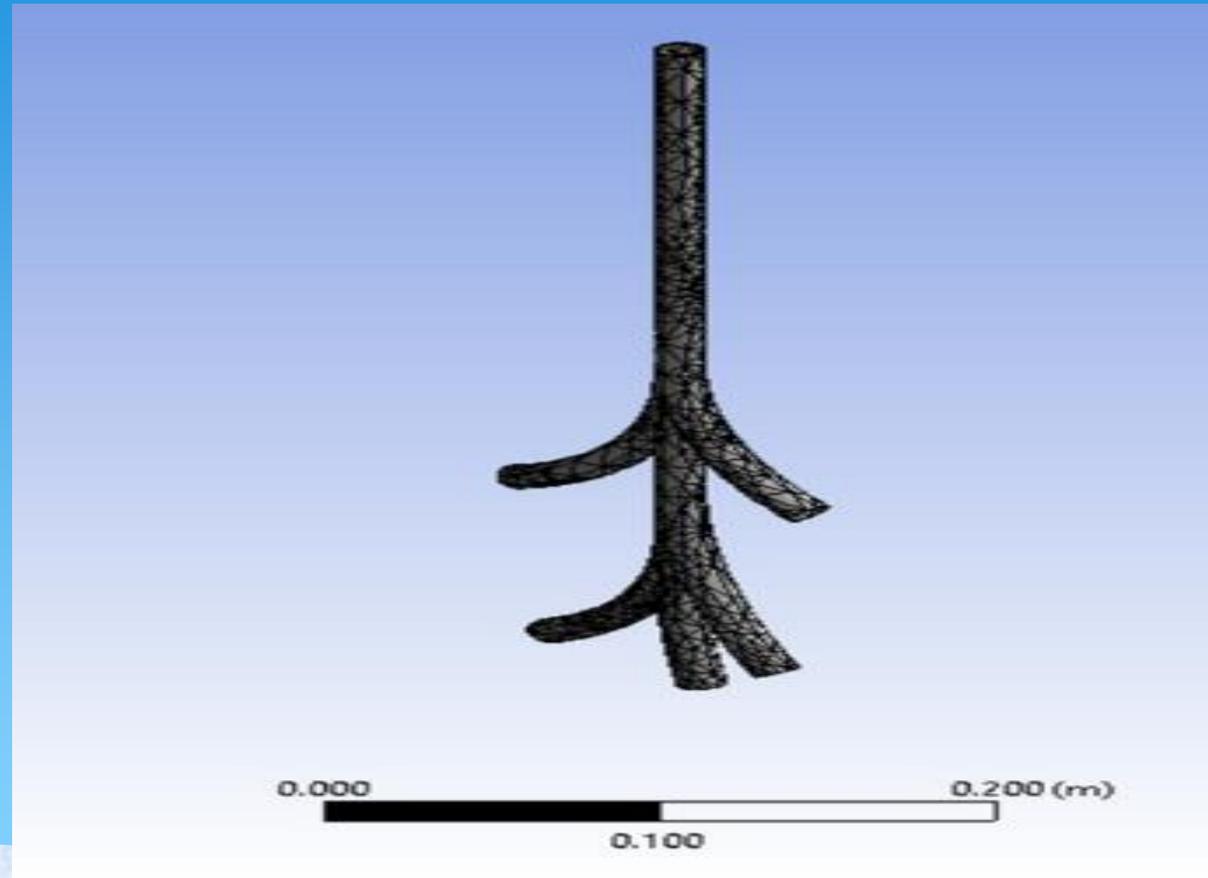
### **CAD Modelling**

#### **2.2 Meshing and Numerical Simulation Setup**

**The geometries were later imported into the ANSYS software workbench, where mesh was applied and hydrostatic pressure settings were set. An element size of 0.5 was imposed, which yielded a total of 3545; 3152 and 3177; 3077 nodes and number of elements as shown in Figure 2.**

# Methodology

## CAD Modelling



**Figure 2. Modal mesh of the arterial vessel**

## **Methodology**

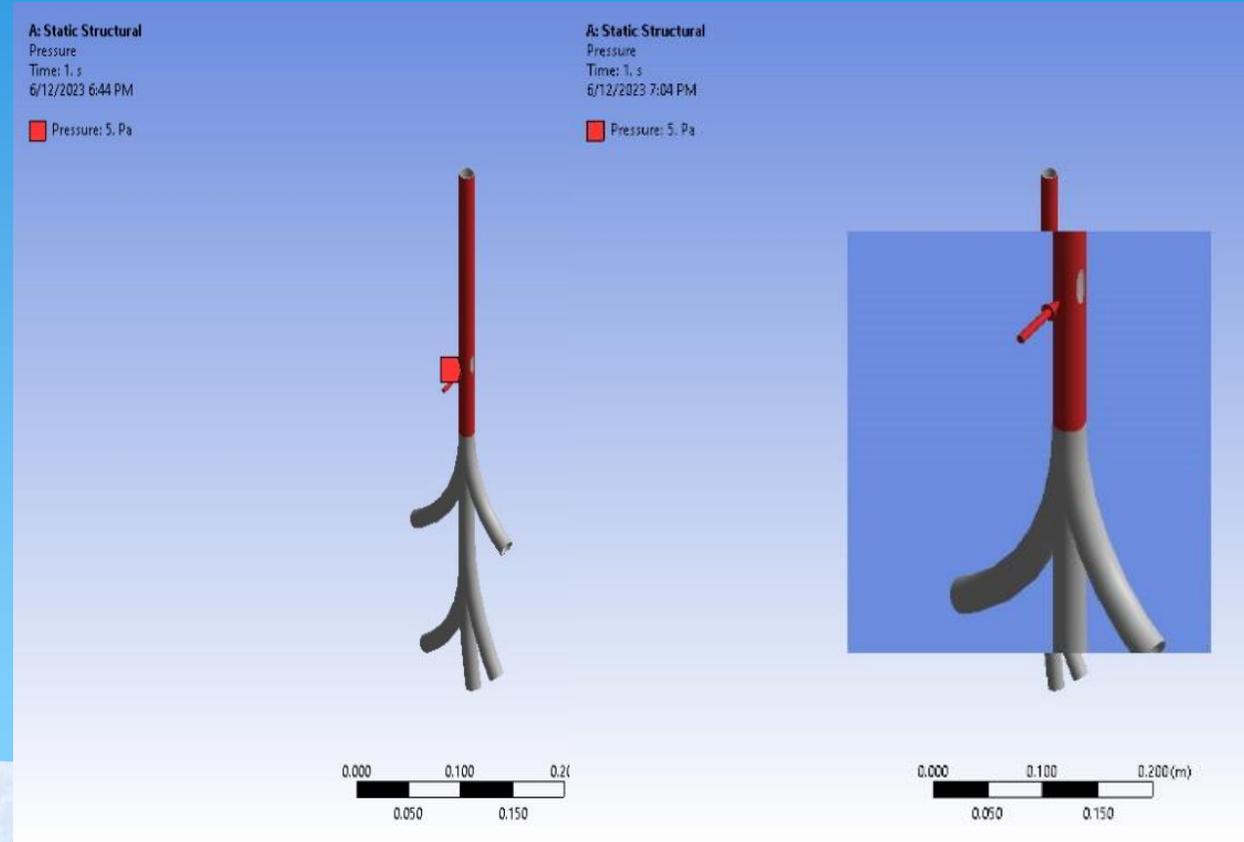
### **CAD Modelling**

#### **2.2 Meshing and Numerical Simulation Setup**

**The muscle direction was imposed on the arterial vessel wall through directional pressure of 5Pa, shown in Figure 3.**

# Methodology

## CAD Modelling



**Figure 3. Flow setup in model by applying pressure of 5 Pa (top) and visual view of the 5 Pa pressure exceeded on the arterial wall by the muscle (below)**

# **Methodology**

## **CAD Modelling**

### **Assumptions and Governing Equations**

#### **2.3.1 Assumptions**

**The basic assumptions made in this study are:**

- i. The body is flexible;**
- ii. There is no slip at the wall;**
- iii. The fluid is incompressible**

# Methodology

## CAD Modelling

### Assumptions and Governing Equations

#### Governing Equations

For a system such as this, Newton's second law is utilized, described in Eqn. 1 and 2.

$$\rho \left( \frac{\partial v_r}{\partial t} + v_r \frac{\partial v_r}{\partial r} + \frac{v_\theta}{r} \frac{\partial v_r}{\partial \theta} - \frac{v_\theta^2}{r} + v_z \frac{\partial v_r}{\partial z} \right) \quad (1)$$

The derivation for acceleration of the blood flow during muscle contraction becomes

$$= \rho g_r - \frac{\partial p}{\partial z} + \mu \left[ \frac{\partial}{\partial r} \left( \frac{1}{r} \frac{\partial}{\partial r} (r v_r) \right) + \frac{1}{r^2} \frac{\partial^2 v_r}{\partial \theta^2} - \frac{2}{r^2} \frac{\partial v_\theta}{\partial \theta} + \frac{\partial^2 v_r}{\partial z^2} \right] \quad (2)$$

$$= \rho g_z \frac{\partial p}{\partial z} + \mu \left[ \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial v_z}{\partial r} \right) + \frac{1}{r^2} \frac{\partial^2 v_z}{\partial \theta^2} + \frac{\partial^2 v_z}{\partial z^2} \right] \quad (3)$$

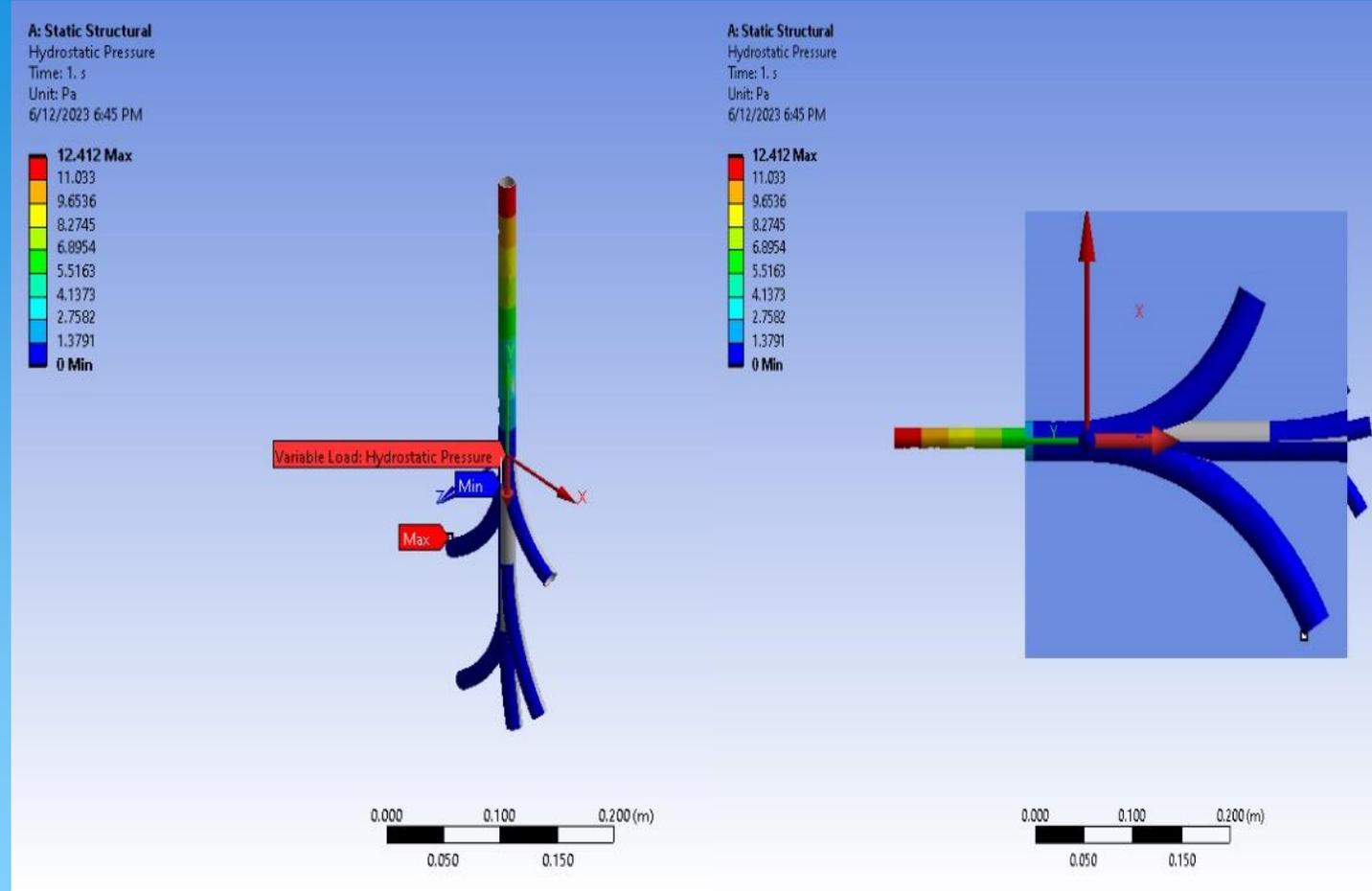
$$= \rho \left( \frac{\partial v_\theta}{\partial t} + v_r \frac{\partial v_\theta}{\partial r} + \frac{v_\theta}{r} \frac{\partial v_\theta}{\partial \theta} + \frac{v_r v_\theta}{r} + v_z \frac{\partial v_\theta}{\partial z} \right) \quad (4)$$

$$= \rho g_\theta - \frac{\partial p}{\partial \theta} + \mu \left[ \frac{\partial}{\partial r} \left( \frac{1}{r} \frac{\partial}{\partial r} (r v_\theta) \right) + \frac{1}{r^2} \frac{\partial^2 v_\theta}{\partial \theta^2} - \frac{2}{r^2} \frac{\partial v_r}{\partial \theta} + \frac{\partial^2 v_\theta}{\partial z^2} \right] \quad (5)$$

## Results and Discussion

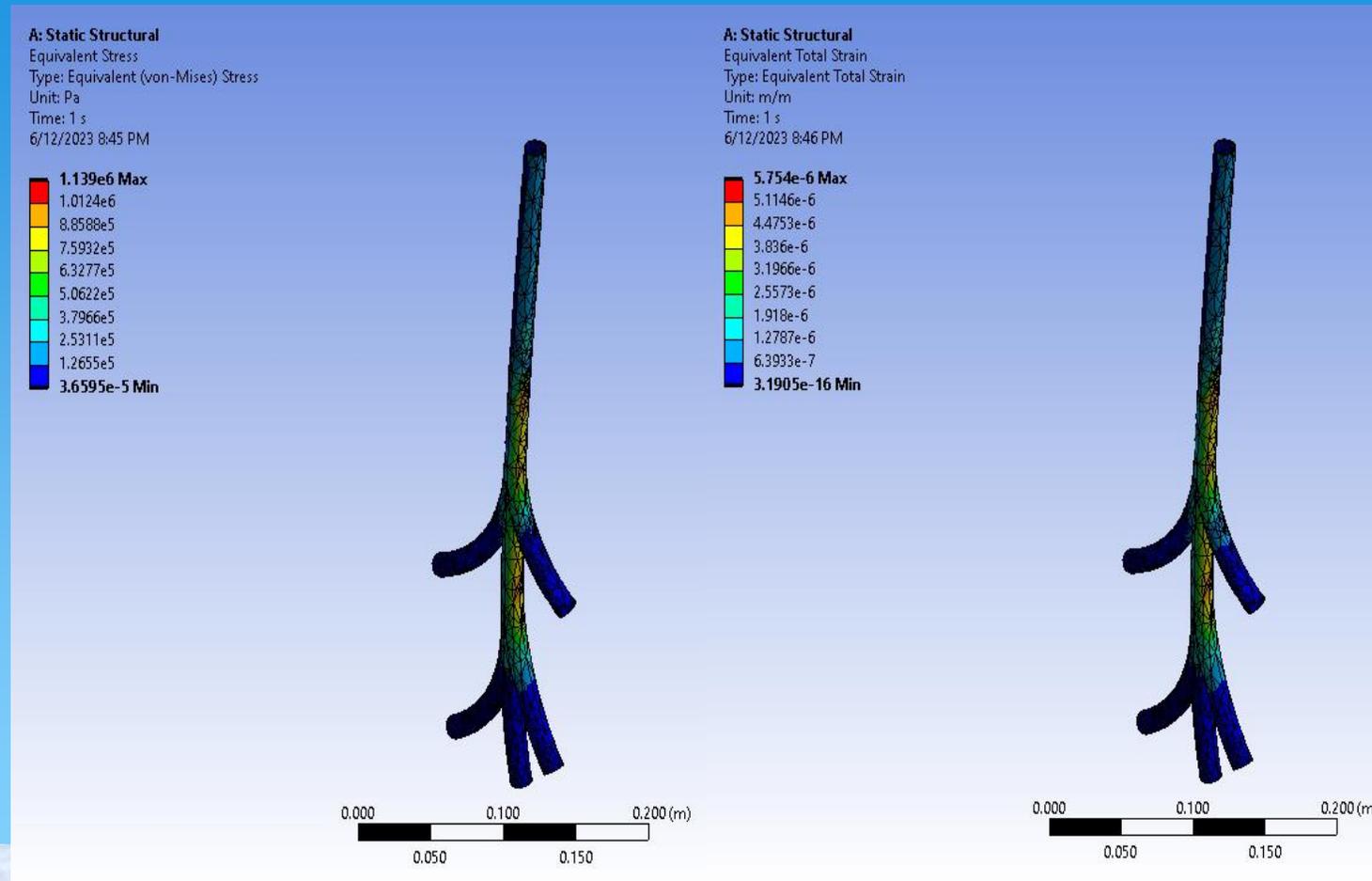
The simulation results provide information about effect of hydrostatic pressure on the arterial wall by the contraction of the muscle, providing insights into the mechanisms underlying high blood pressure. The results in both cases under consideration are presented in Figures 6-14.

# Results and Discussion



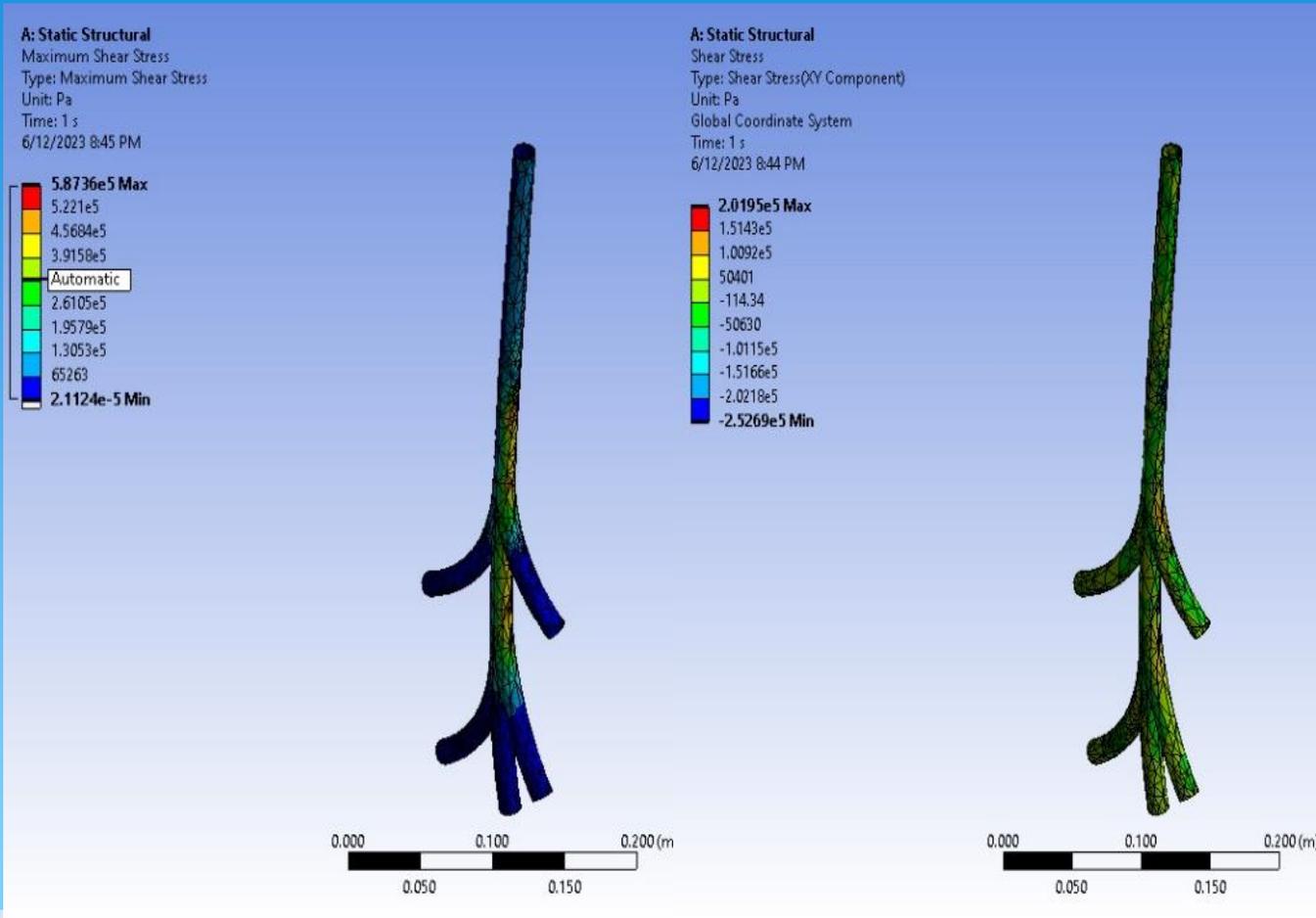
**Figure 6. Hydrostatic pressure at minimum and maximum (left) and virtual view of the hydrostatic pressure (right), resulting from static structural analysis**

# Results and Discussion



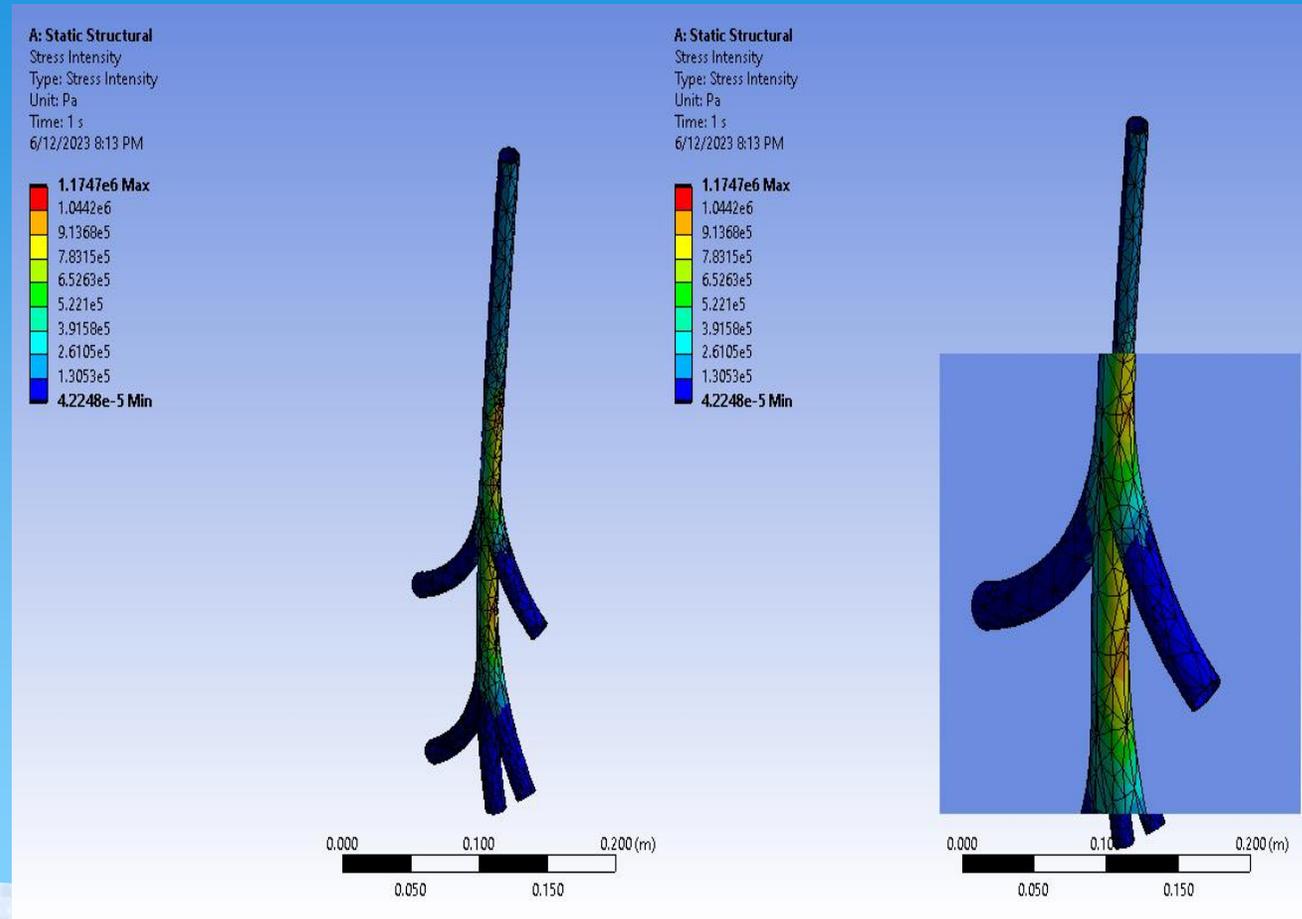
**Figure 7. Equivalent stress acting on the model (left) and region of equivalent total strain stress (right) during static condition**

# Results and Discussion



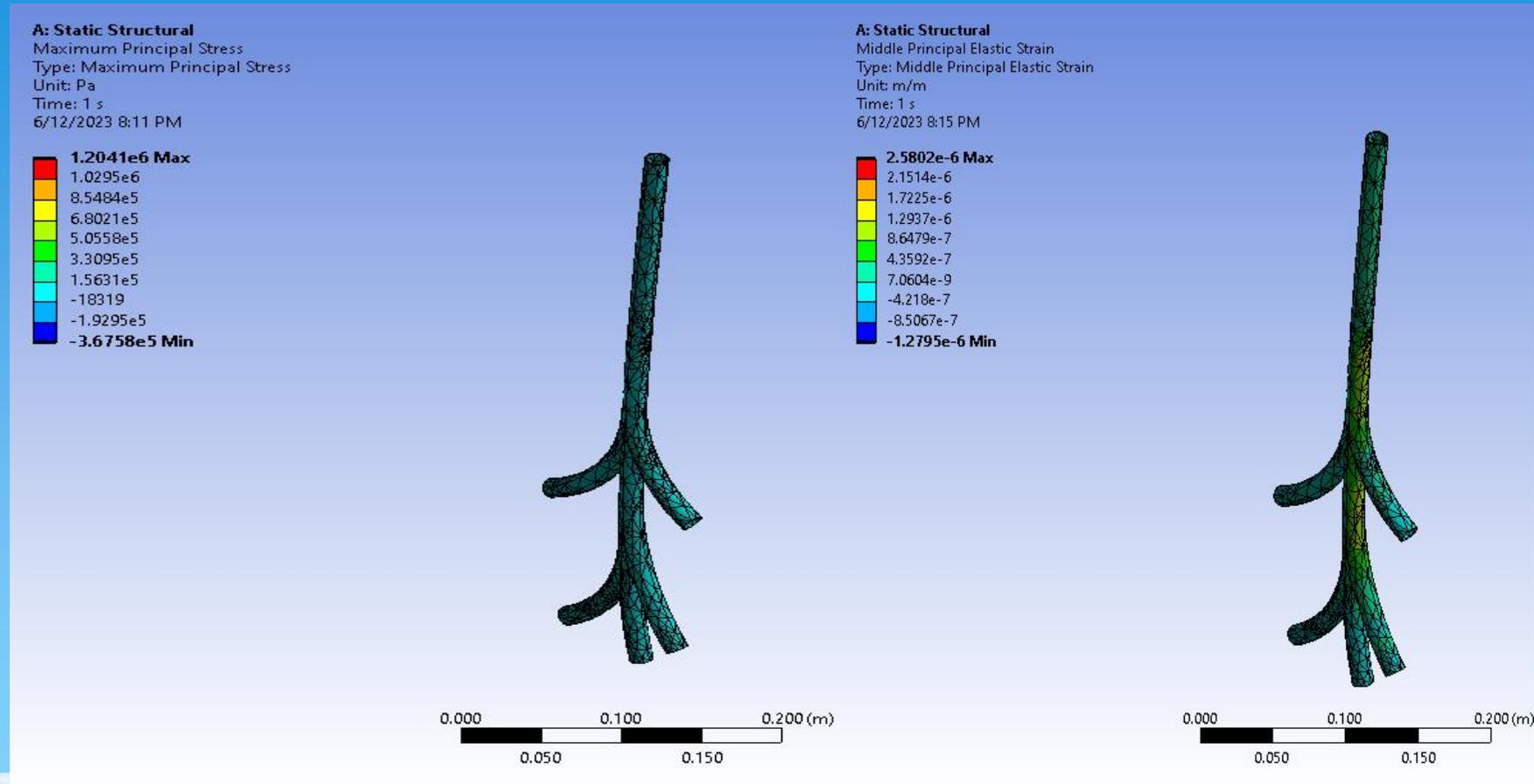
**Figure 8. Effect of maximum shear stress on the wall (left) and shear stress on the wall by the muscular force (right) during static structural condition**

# Results and Discussion



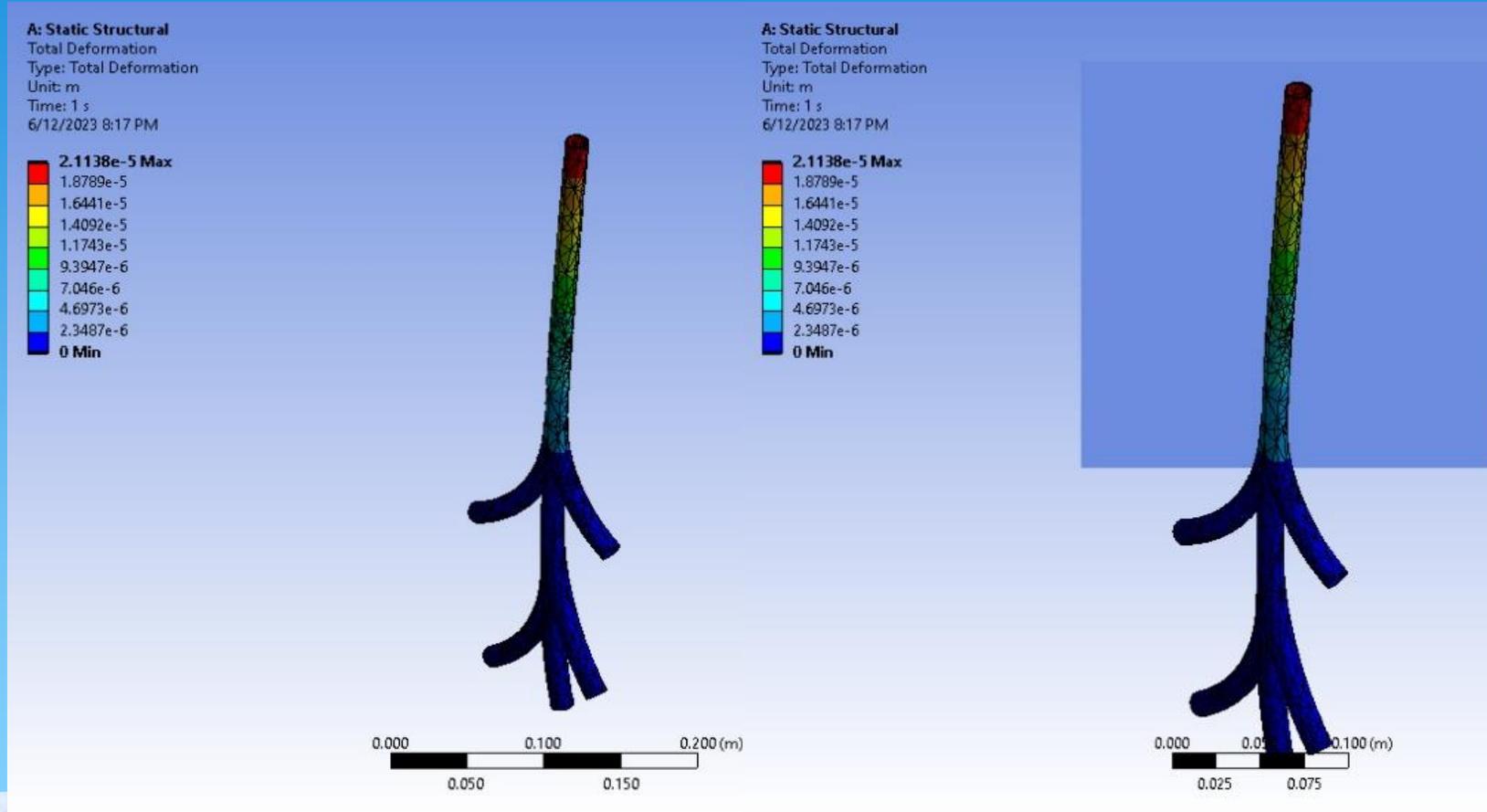
**Figure 9. Stress intensity behaviour across the model (left) and elastic strain resulting from the hydrostatic pressure during static condition (right)**

# Results and Discussion



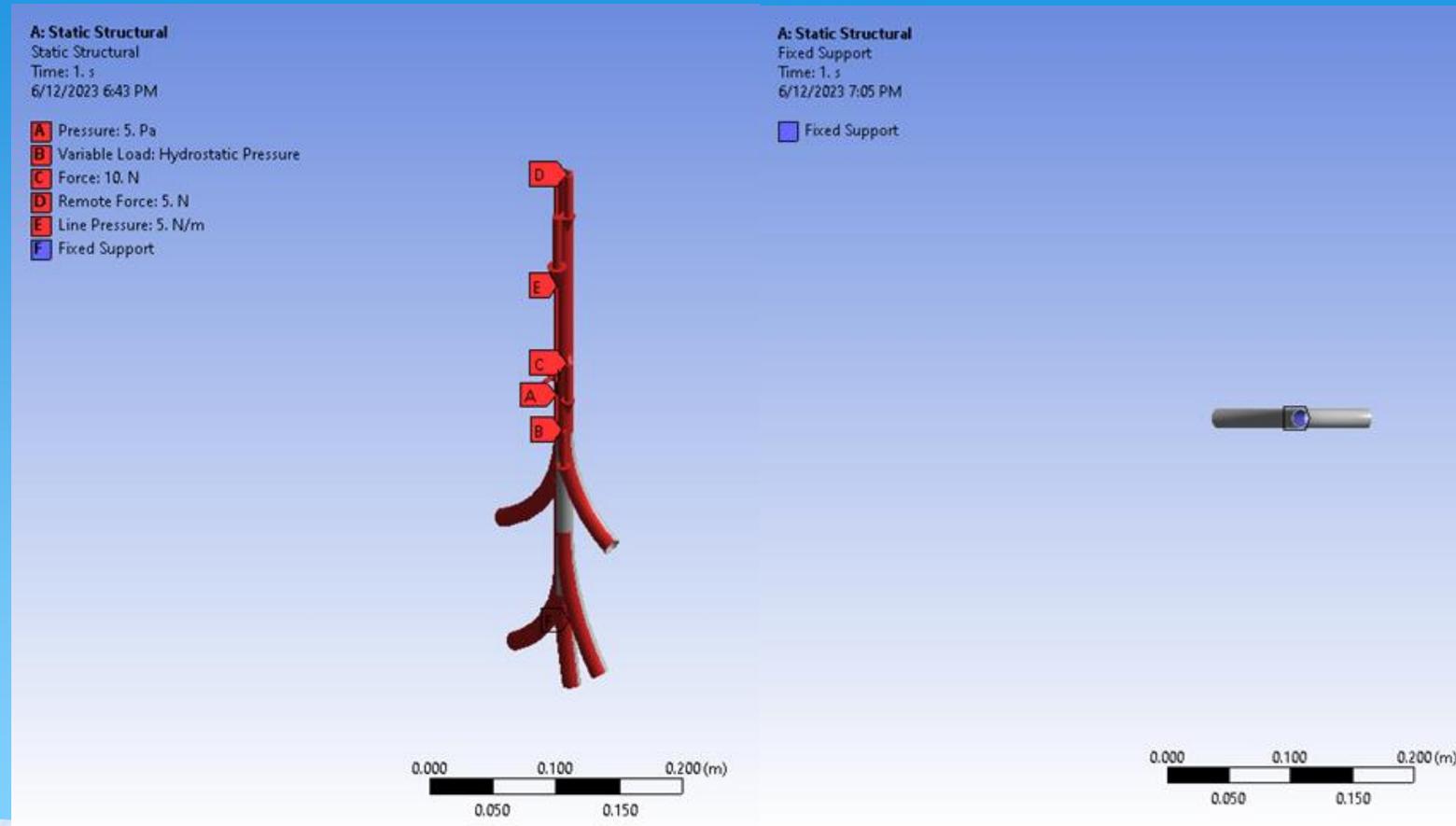
**Figure 10. Maximum principal stress on the model (left) and region of middle principal elastic strain (right) during static condition**

# Results and Discussion



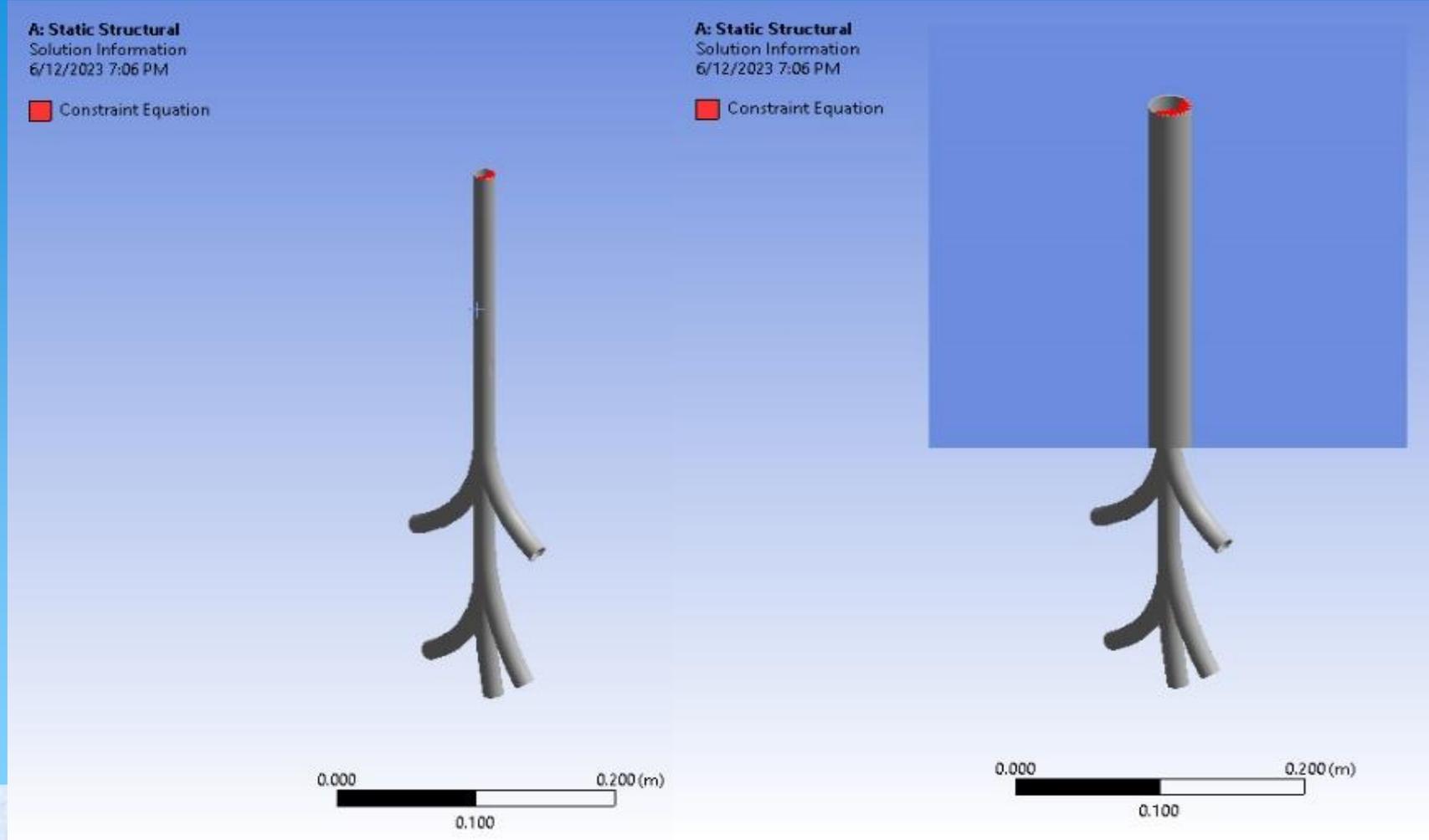
**Figure 11. Total deformation obtained on the model (left) and region of maximum stress (right) during elongation**

# Results and Discussion



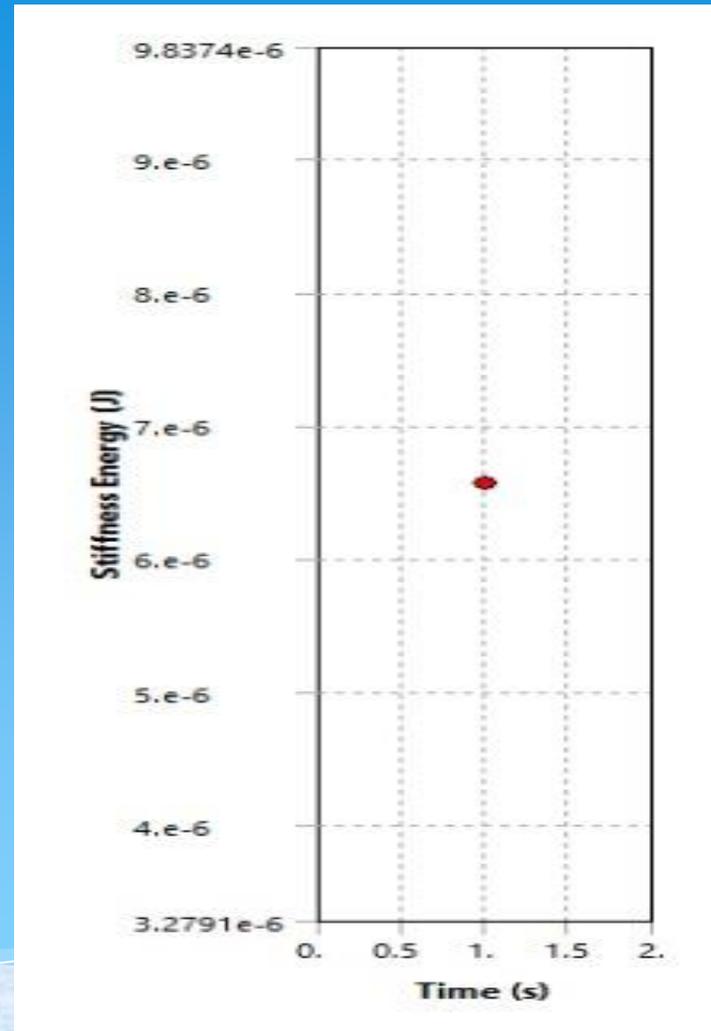
**Figure 12. Overall stress on the model showing pressure, variable load, force, remote force and fixed support(left) and area region of fixed support (right)**

# Results and Discussion



**Figure 13. Blood flow pattern resulting from the same hemodynamic pressure during contraction of the muscle and arterial blood vessel (left) and visible view of the blood flow (right)**

## Results and Discussion



**Figure 14. Blood flow pattern resulting from the same hydrostatic pressure during flaccidity**

# DISCUSSION OF RESULTS

**As seen in the figures, the results of this study suggest several clinical implications regarding the hemodynamic factors affecting high blood pressure should not exceed stiffness energy of about  $6.6e-6$  in 1 second. First, the observation of extreme total deformation at the point of load application during static condition (see Fig. 14) indicates a potential structural weakness in the arterial blood vessel. This could be associated with the compromised integrity of the vessel, such as reduced elasticity and fibrosis, commonly observed in arterial diseases and dysfunctions, such as atherosclerosis, aneurysms, and arterial stiffness. These findings highlight the importance of assessing and addressing structural abnormalities in patients with arterial diseases and dysfunctions, as they may contribute to the inadequate rigidity necessary for passage of blood through the arterial wall**

# Conclusions

**The studies provided fundamental understanding of the role of hemodynamic factor during high blood pressure and mechanical processes involving large muscle contraction on the arterial wall, using computational approach. The findings emphasize the importance of evaluation of the result of various interventions or treatments targeting high blood pressure and guide the development of novel therapeutic approaches. Future research and clinical interventions aimed at improving arterial vessel integrity, enhancing blood flow and addressing high blood pressure may lead to more effective management strategies for individuals with high blood pressure. Applications of computational modeling of arterial blood vessels will help the study of atherosclerosis development, which will analyze the effects of stenting or bypass surgeries. These will help investigate the impact of arterial stiffness on blood flow, and optimizing treatment strategies for various cardiovascular diseases.**

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