Technical Memorandum 0315105
7 March 2023

Evaluation of the Surgical Intervention Point for Carotid Artery Bifurcation Stenosis through Blood Flow Simulation

Executive Summary

This technical memorandum analyzes blood flow parameters at the carotid artery bifurcation to determine the parameters for recommending medical intervention in the presence of blockages. The evaluation models various degrees of obstruction in the internal carotid artery (ICA), and in both the ICA and the external carotid artery (ECA). These blockages imitate stenosis ranging from 50% to 80% and highlight the primary concern of this report: assessing the concerns of carotid artery disease and atherosclerosis.

In order to measure the effects of plaque formation due to stenosis, SOLIDWORKS Flow Simulation was used to conduct the analysis of wall shear stress, blood velocity, shear rate, and outlet mass flow rate based on the varying stenosis levels. Results were thus derived from the comparison of fluid mechanical parameters in the obstructed arteries with an unobstructed baseline artery. The results were found to be in disagreement with the established values from prior studies within the literature, but independent conclusions from the data analysis suggested that doctors should surgically intervene when stenosis levels reach 70% occlusion.

Introduction

Many life-threatening medical concerns such as stroke, transient ischemic attack (TIA), and loss of vision can be correlated to the occurrence of atherosclerosis blockages within the internal and external carotid arteries. The ICA acts as the primary blood supplier to the brain, and the ECA to the face and extracranial structures.[1] In terms of mitigating these concerns, there are several factors that might affect the need for surgical intervention, including genetic factors, lifestyle choices, and underlying health conditions. This report will hone in on the mechanical state of the carotid artery bifurcation in order to identify the degree of arterial occlusion at which surgical intervention should be considered. The current convention for surgical intervention, according to the Journal of the American Heart Association, suggests that carotid endarterectomy (CEA) be performed at severe stenosis levels, where diameter reduction is greater than or equal to 70%. As a highly invasive surgery, CEA must be operated under the discretion of the surgery team, taking severity and frequency of symptoms, as well as underlying medical conditions, into account.[2] As the most reliable recommendation for stroke prevention, the results of this study should independently confirm or deny its validity.

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[1] Ogoh, Shigehiko. “Regional Redistribution of Blood Flow in the External and Internal Carotid Arteries During Acute Hypotension”. Regulatory, Integrative and Comparative Physiology, May 15, 2014.

[2] Meschia, James F. “Guidelines for the Primary Prevention of Stroke | Stroke.” Stroke. American Heart Association Stroke Council, October 28, 2014.

Within the literature, two measured parameters stand out as telltale indicators of a potential carotid artery disease emergency: arterial wall shear stress (WSS), and outlet blood flow velocity. According to carotid artery fluid dynamic analyses by Filardi, a healthy ICA should exhibit a WSS of around 83 Pa. In an ICA suffering from 30% stenosis, it was shown that the maximum WSS approaches a peak of nearly 360 Pa at its throat. While the stenosis is only a degree of 30%, these values may ensue danger, as the stenosed artery has a value promoting endothelial cell damage. As arteries become more stenosed, the WSS values should increase and thus the likelihood of arterial damage.[3] According to another study linking carotid blood flow geometry to disease by Nguyen et al., a WSS value of 0.4 Pa serves as the threshold between atherogenesis, and healthy arterial walls and blood flow.[4] Together, these studies establish a general range of healthy WSS values. With regards to blood velocity, Filardi's study finds that the peak value for a healthy artery shows to be about 2.45 m/s, while the peak velocity occurring at the throat of stenosis is slightly over double, 5.06 m/s.[5] To sustain constant blood flow rate, blood velocity will increase as it approaches a narrowed artery. However, in the presence of another path, such as the carotid bifurcation, blood can divert from a stenosed artery to flow through a healthy counterpart.

Fluid flow changes correlated to these two parameters have direct biological implications. The local effects of plaque formation due to stenosed arteries lead to turbulent blood flow and increased stress on arterial walls.[6] As blood interacts with plaqued arteries, it flows over the blockages and becomes turbulent, causing the arterial walls to destabilize; this destabilization is a consequence of increased WSS and may occlude the blood supply to the brain.[7,8] Holistically, this report looks towards outlet blood flow velocity to indicate the rate of oxygen-rich blood flow to the brain. The outflow of oxygenated blood decreases as atherosclerosis becomes symptomatic in the ICA and leads to strokes or TIA.[9] Because of the narrowing of the arteries, the blood flow velocity to the brain becomes limited. Therefore, it is crucial to consider the WSS and blood flow

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[3] Filardi, V. "Carotid Artery Stenosis near a Bifurcation Investigated by Fluid Dynamic Analyses." The Neuroradiology Journal. U.S. National Library of Medicine, August 2013.

[4] Nguyen, Kien T, R. Stokholm, R.F. Smith, A. Poli, K. Perktold, M. Nazemi, J.M. Laurberg, D.N. Ku, M. Kato, and F.J.H. Gijsen. "Carotid Geometry Effects on Blood Flow and on Risk for Vascular Disease." Journal of Biomechanics. Elsevier, October 4, 2007.

[5] Filardi, V. The Neuroradiology Journal. 2013

[6] Jahangiri, Mehdi, Mohsen Saghafian, and Mahmood Reza Sadeghi. "Numerical Study of Turbulent Pulsatile Blood Flow through Stenosed Artery Using Fluid-Solid Interaction." Computational and mathematical methods in medicine. U.S. National Library of Medicine, 2015.

[7] R;, Khanafer KM;Bull JL;Upchurch GR;Berguer. "Turbulence Significantly Increases Pressure and Fluid Shear Stress in an Aortic Aneurysm Model under Resting and Exercise Flow Conditions." Annals of vascular surgery. U.S. National Library of Medicine, January 21, 2007.

[8] Filardi, V. The Neuroradiology Journal. 2013.

[9] "Carotid Artery Disease." Mayo Clinic. Mayo Foundation for Medical Education and Research, October 4, 2018.

velocity after stenosis in the carotid artery when making recommendations, as these parameters have direct associations with significant medical impacts.

The methodology used to investigate stenosed arterial blood output and arterial shear was directed by the parameters provided in the 214 Biomedical Request for Information. The study was conducted in two phases, initially modeling blockages in the interior arterial branch followed by those in both the interior and exterior carotid arteries. The collected data includes computationally generated cut plots illustrating velocity and shear rate, xy plots mapping blood flow with respect to shear rate (1/s) over length (m), and surface parameter analysis comparing bulk average velocities. The purpose of this technical memo is to demonstrate SOLIDWORKS simulations of the measured points of arterial occlusion that result in dangerous values of WSS and velocity. The aforementioned case simulations confirm the degree of arterial occlusion where surgical intervention is recommended due to high risk conditions within the carotid artery.

Results

Note: Each figure referenced within the Attachments section below will use A-E labeling, representing the visualization for diameter stenosis of degrees 0%, 50%, 60%, 70%, and 80% respectively.

From the simulations conducted, the most significant result, to first note, is that the simulated values agree with few to none of the values stated in either of the cited literature sources. This recurrent theme of deviation of magnitude will be mentioned and analyzed throughout the analysis of results. Without a firmly established basis of magnitude to use as a cutoff point for many of the simulations conducted, the major conclusions and recommendations for surgical interventions will have to be based on the relations between variables and noteworthy trends within the simulations. The results discovered indicated that these analysis outcomes are largely independent of either of the prior studies conducted.

Outlet Blood Velocity in Stenosed ICA

For the outlet velocity values at the solely ICA stenosed arteries, at no point do the values approach the 2.45 m/s (healthy) or 5.06 m/s (dangerous) velocity values found within Filardi's study. This handicaps the usefulness of the outlet velocities purely in a data vacuum, as they are shown in Table 1.

However, a notable trend does occur based on the velocity models in Figure 1. While the simulations are inconclusive on the exact magnitude demanding intervention, a definitive claim can be made with absolute certainty: a distributed area of 0 m/s flow sufficient to fully saturate a section of an artery, indicating a lethal carotid blockage, is unacceptable for any patient. Here, Figure 1 proves useful: large, impassible regions of heavy blue (indicating a 0 m/s velocity, or total blockage being very likely to occur) clearly begin to appear at the inlet and outlet both of Figure 1D. Figures 1B and 1C have segments gradually becoming more blue, a cause for

significant concern; but a potentially fatal level of blockage does not begin to develop until around 70%. Thus, based solely off of the ICA velocity measurements, 70% stenosis is where medical professionals should consider urgent intervention.

Stenosis	Branch	Outlet Velocity Bulk Average (m/s)
0%	ICA	0.282
0%	ECA	0.282
50%	ICA	0.172
0%	ECA	0.433
60%	ICA	0.107
0%	ECA	0.475
70%	ICA	0.04
0%	ECA	0.519
80%	ICA	0.004
0%	ECA	0.548

Table 1: *Outlet Velocities at Differing Stenosis Levels Within the ICA only.*

Outlet Blood Velocity in Coincidentally Stenosed ICA and ECA

Initially, the magnitude values found in Figure 2, especially going up to the 80% stenosis model shown in Figure 2.E., may be seen approaching the 5.06 m/s danger values found within Filardi's study. However, Filardi's findings were reported based on the numerical values of a model that had stenosis in one side of the carotid artery split; since Figure 2 is simulating both ICA and ECA stenosis, this means that the exact magnitude of the data within the literature found is not applicable to this study, as was the case with the solely ICA-stenosed simulations.

The distribution of velocities shown in the different models will guide the analysis outcome. Similar to the prior ICA-stenosed simulation, the renders of ICA and ECA stenosis in Figure 2 demonstrate a noticeable decrease in velocity flow as stenosis progresses; but it is again not until the 70% mark in Figure 2.D. that definitively lethal blockages of 0 m/s begin to dominate the artery both before and after the stenoses. Once more, the results of the velocity flow rate simulation favor an urgent intervention recommendation at 70% stenosis within both the ICA and ECA.

Stenosis	Branch	Outlet Velocity Bulk Average (m/s)
0%	ICA, ECA	0.282
50%	ICA, ECA	0.398
60%	ICA, ECA	0.45
70%	ICA, ECA	0.41
80%	ICA, ECA	0.6045

Table 2: *Outlet Velocities at Differing Stenosis Levels Within ICA/ECA Double Stenosed Models.*

Shear Stress in Stenosed ICA

While the velocity flow rate simulations discussed previously are vital to consider when intervening to save a patient, the literature discussed in the introduction points to the idea that shear stress is the data most relevant to the local destruction of endothelial tissue in the event of stenosis occurring. This places the focal point of the discussion for WSS around the dangers to the local tissue based on the WSS values present.

Much like with the velocity values, the shear stress values discovered within the simulations did not come close to the values given within the literature. Nguyen et al. identified 0.4 Pa as being a dangerous minimum value, below which plaque begins to undergo atherogenesis, likely due to lack of sufficient shear stress to keep the plaque broken and moving without adhering to the arterial walls. Across all of the shear stress models demonstrated in this document, the absolute lowest WSS value, 0.4 Pa, occurred at exactly 0% stenosis in Table 3. This was recorded overwhelmingly closer to the middle of the artery as opposed to the wall shear stress of interest, as can be seen in the shear stress distributions in Figure 3, where the teals of higher shear rates only began to occur in the occlusion or at the edges of the artery. As the value never goes below 0.4 Pa once, only increasing across models, the 0.4 Pa minimum danger cutoff was not useful in finding a surgical recommendation.

Filardi's study places 83 Pa as the shear stress of a fully unstenosed carotid artery. Filardi then specifically identifies the peak stenosis throat WSS value of 360 Pa as the point of greatest concern. The data within the simulations here reach a maximum value of a mere 10.56 Pa at the absolute peak, shown in table 3; this disagrees significantly with the magnitude of Filardi's WSS values. While the WSS magnitudes simulated here may deviate from literature, the useful takeaway is the method of identifying a peak WSS value and basing the awareness of dangerous shear stress around the maximum across all of the simulations. Within the data here, a noticeable trend occurs: the stenosis throat WSS increases rapidly until 70%, after which it begins to quickly fall off as the stenosis increases further past 70%.

This is unusual. By conventional logic, the WSS should continue to increase up to 100% as the stenosis tightens the blood flow further. However, the simulations, as shown in the peak generated values of Table 3, clearly show that 70% stenosis represents the percentage model at which the highest and most damaging shear stress of 10.56 Pa is present. The only reasonable explanation for this is that 70% stenosis simply represents the point at which the geometries of the stenosis, perhaps due to the slope of the tissue curvature or the angle of fluid approach leading up to the constriction, are most affected by the blood flow. This is also likely due to the fact that past 70% constriction in the ICA, the blood flow begins to follow the path of least resistance instead (the unblocked ECA), rather than the over constricted ICA.

While the magnitude of WSS is inconclusive, regardless, Figure 3, Figure 4, and Table 3 all demonstrate that a 70% ICA blockage represents the point at which medical intervention becomes most dire. Just as Filardi concluded, this is on account of the fact that the maximum

WSS value is where the endothelial tissue is subjected to the most damage. It is notable that the maximum shear stress values occur within the same models where 0 m/s blood velocity begins to block the ICA blood flow completely; this may be taken as further indication of the relevance of the 70% stenosis value specifically being the norm for recommending intervention.

Stenosis in ICA	Maximum Shear Rate at Throat of Stenosis, ~ 0.070 m (1/s)	Blood Viscocity (Pa*s)	Shear Stress (Pa)
0%	100	0.004	0.4
50%	800	0.004	3.2
60%	1750	0.004	7
70%	2640	0.004	10.56
80%	1500	0.004	6

Table 3: *Shear Rate Data for stenosis in the ICA. Note the peak value at 70% before the dropoff at percentages afterwards. Blood viscosity values were found to be approximately 4.0 cP, thus shear stress values were calculated using the relationship: shear stress = shear rate * blood viscosity.* [10,11]

Shear Stress in Coincidentally Stenosed ICA and ECA

The results from Figure 5 and Table 4 are, in an informational vacuum, inconclusive. Unlike the ICA stenosis shear stress models, there is no path of least resistance to take, as both outlets from the carotid artery are equally constricted. The data shows a standard increase of WSS from 0% to 100%, and without definitive literature values, lends no analysis. However, in the ICA stenosis models above, 10.56 Pa is noted as the maximum WSS value, which can be considered the magnitude at which the WSS becomes unacceptable. Caution should be exercised when using results from one model to define the dangerous values for another model, as it may not always be appropriate to set maximum tolerance based solely on these results. As the focal point of evaluating wall shear stress in carotid artery stenosis is to determine the threshold at which endothelial cell damage occurs, though, safety precautions must be taken to prevent progression beyond that point in the patient's artery. Using that logic, interpolation in Table 4 paired with visuals from Figure 5 show that this 10.56 Pa value is passed between the 50% and 60% models.

This would indicate from the ICA/ECA stenosis shear stress model that 50% is the recommended degree of stenosis for surgical intervention, before the wall shear stress surpasses the maximum tolerance defined in the ICA stenosis models.

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[10] LoGerfo, Frank W, Michael D Nowak, William C Quist, Howard M Crawshaw, and Bala K Bharadvaj. “Flow Studies in a Model Carotid Bifurcation.” ahajournals. ahajournals, 1981.

[11] Tremblay, Joshua C. “Through thick and thin: The interdependence of blood viscosity, shear stress and endothelial function” Experimental Physiology. The Physiological Society, December 6, 2019.

Stenosis in ICA + ECA	Maximum Shear Rate at Throat of Stenosis, ~ 0.070 m (1/s)	Blood Viscocity (Pa*s)	Shear Stress (Pa)
0%	100	0.004	0.4
50%	1800	0.004	7.2
60%	6300	0.004	25.2
70%	25200	0.004	100.8
80%	70400	0.004	281.6

Table 4: *Shear Rate Data for stenosis in both the ECA and ICA. The shear stress = shear rate * blood viscosity relationship used in Table 2 is observed in Table 3.*

Conclusion

The decision for surgical intervention on stenosed arteries is based on various factors, including the degree of stenosis, the presence of symptoms, and the overall health of the patient. According to the American Heart Association and the American Stroke Association, surgical intervention is generally recommended for patients with symptomatic stenosis of 70% or greater, and for some high-risk asymptomatic patients with 60% to 99% stenosis. Accordingly, the decision for surgical intervention should be made on a case-by-case basis, taking into account individual patient factors and preferences. The velocity and shear stress data within this document both support varying levels of danger in the percentages leading up to 70% stenosis, but assert that surgical intervention should only be strictly necessary at 70% obstruction. Despite its effectiveness, surgery is highly invasive and must be used judiciously; therefore, simulations were used to independently model and verify the degree of stenosis at which less invasive interventions are appropriate and when surgical intervention is necessary.

The simulations disagreed with the previously established velocity and shear stress values within the literature. Independently, in all of the velocity-based simulations due to the presence of 0 m/s cutoffs, and in a shear stress simulation for ICA stenosis due to the occurrence of the maximum shear stress magnitude at the 70% blockage geometries, 70% stenosis was continuously found to be the recommended point of intervention to prevent carotid artery disease development. The only analysis to disagree with the 70% occlusion assessment, the shear stress ICA and ECA stenosis model, only did so if the value from the ICA stenosis shear stress model was stretched to be assumed as a uniformly damaging shear stress value across all models; this is wholly distinct from an established value from another study, and thus puts this single outlier into question. Due to the independent simulations substantiating the previously declared recommendation of 70% by the American Heart Association, this report has concluded that surgical intervention is highly recommended for levels of stenosis at 70% or greater obstruction in ICA as well as ICA and ECA blockage cases.

References

"Carotid Artery Disease." Mayo Clinic. Mayo Foundation for Medical Education and Research, October 4, 2018. https://www.mayoclinic.org/diseases-conditions/carotid-artery-disease/symptoms-causes/syc-20360519#:~:text=Carotid%20artery%20disease%20develops%20slowly,changes%2C%20medication%20and%20sometimes%20surgery.

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R;, Khanafer KM;Bull JL;Upchurch GR;Berguer. "Turbulence Significantly Increases Pressure and Fluid Shear Stress in an Aortic Aneurysm Model under Resting and Exercise Flow Conditions." Annals of vascular surgery. U.S. National Library of Medicine, January 21, 2007. https://pubmed.ncbi.nlm.nih.gov/17349339/#:~:text=Our%20study%20provides%20a%20numerical,increased%20shear%20stress%20in%20aneurysms.

Tremblay, Joshua C. "Through thick and thin: The interdependence of blood viscosity, shear stress and endothelial function" Experimental Physiology. The Physiological Society, December 6, 2019. https://physoc.onlinelibrary.wiley.com/doi/10.1113/EP088315.

Attachments

Note: Each figure uses A-E labeling, representing the visualization for diameter stenosis of degrees 0%, 50%, 60%, 70%, and 80% respectively.

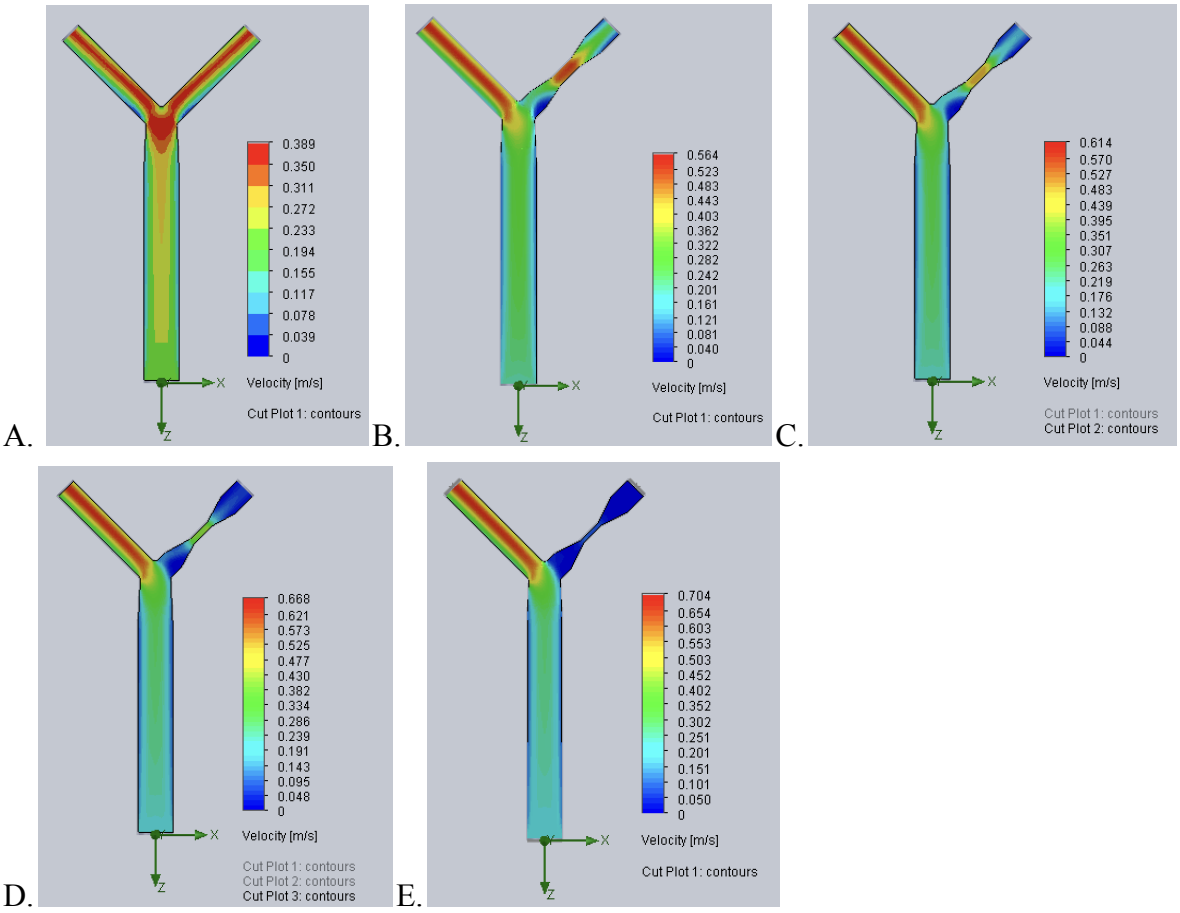
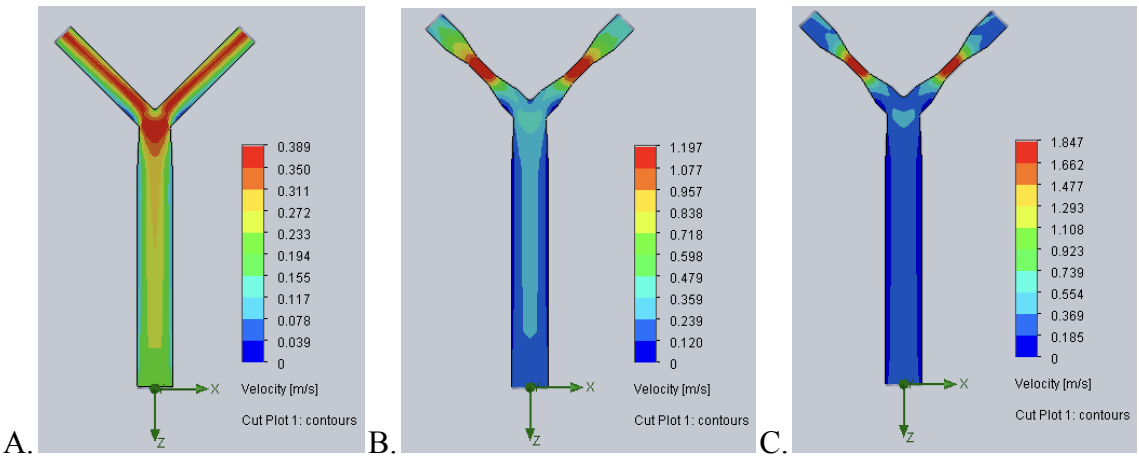
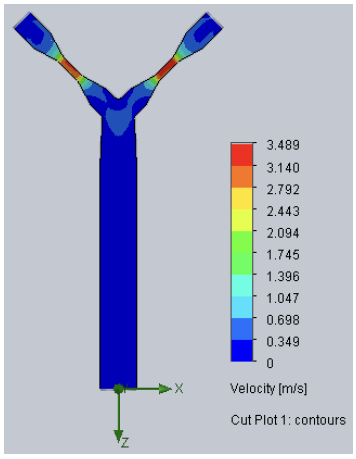


Figure 1: *Velocity cut model showing blood flow velocity distribution at the carotid artery bifurcation with varying degrees of stenosis in the ICA.*



D. 

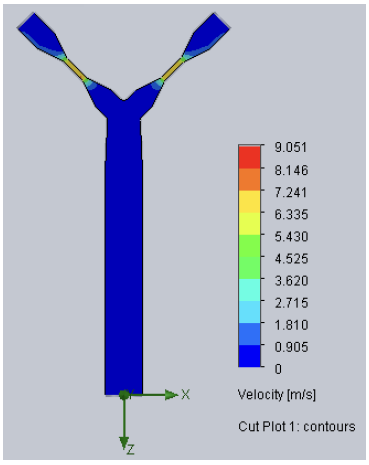
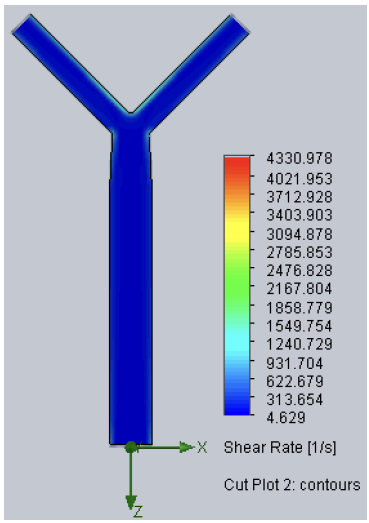
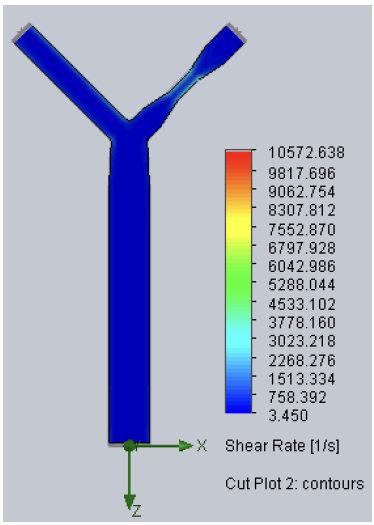
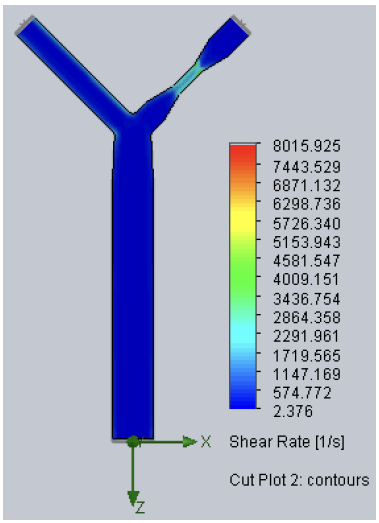
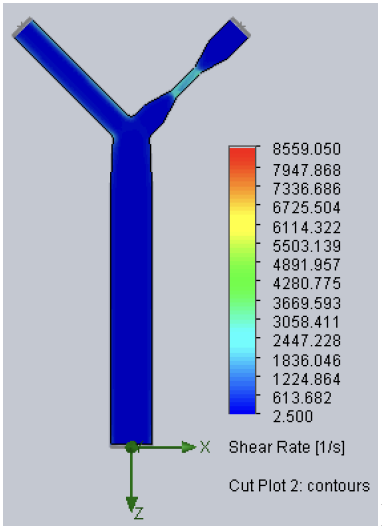
E. 

Figure 2: *Velocity cut model showing blood flow velocity distribution at the carotid artery bifurcation with varying degrees of stenosis in both the ICA and the ECA.*

A. 

B. 

C. 

D. 

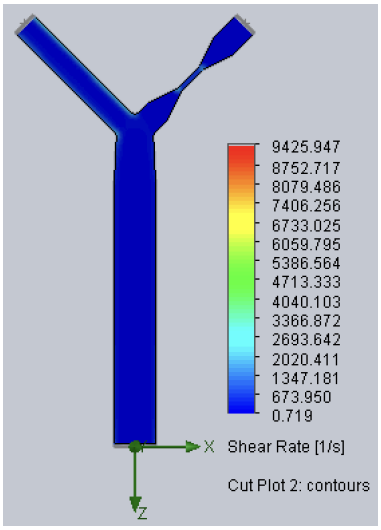
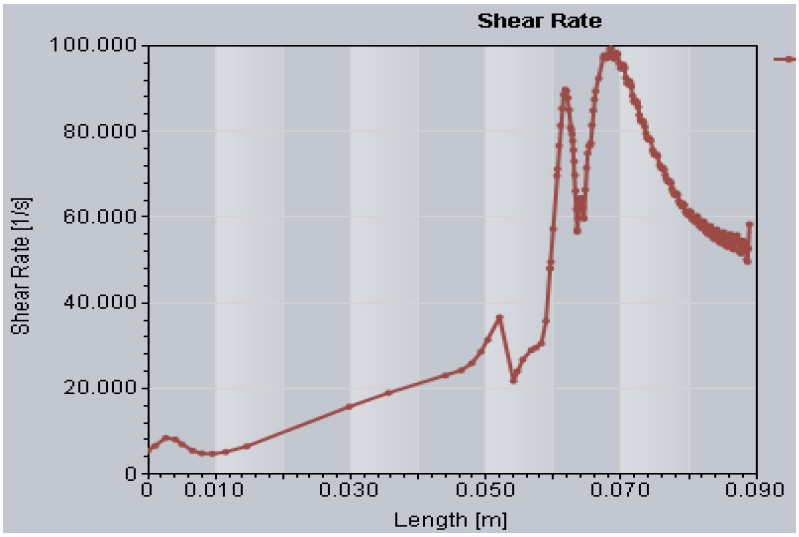
E. 

Figure 3: *Shear rate cut plots showing the changing shear rate at the throat of stenosis in the ICA. Note: Shear rate correlates to shear stress at the same points of interest.*

A. 

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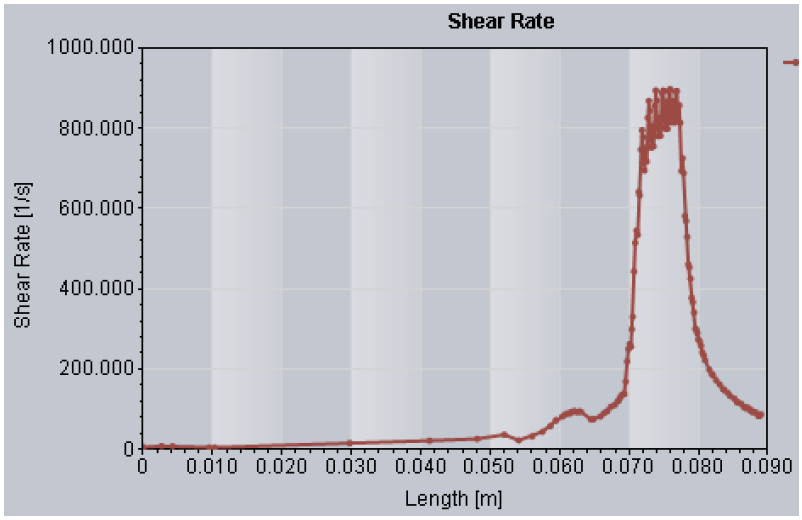
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800.000
600.000
400.000
200.000
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Shear Rate [1/s]
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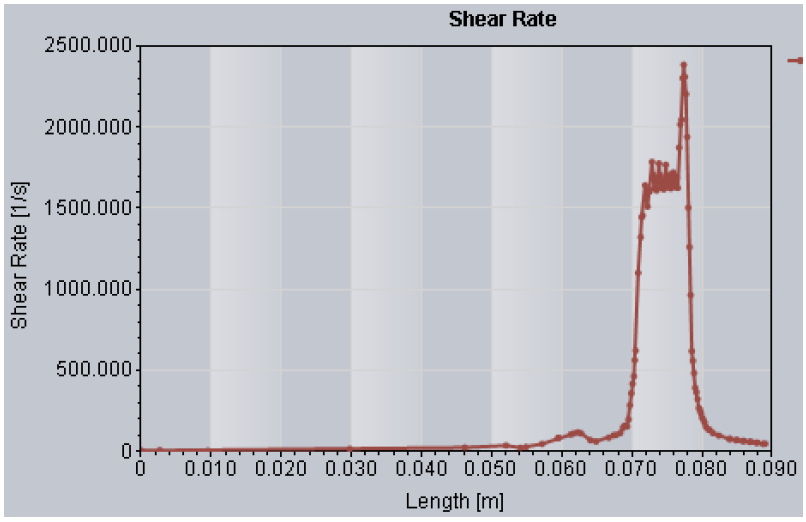
Length [m]

C.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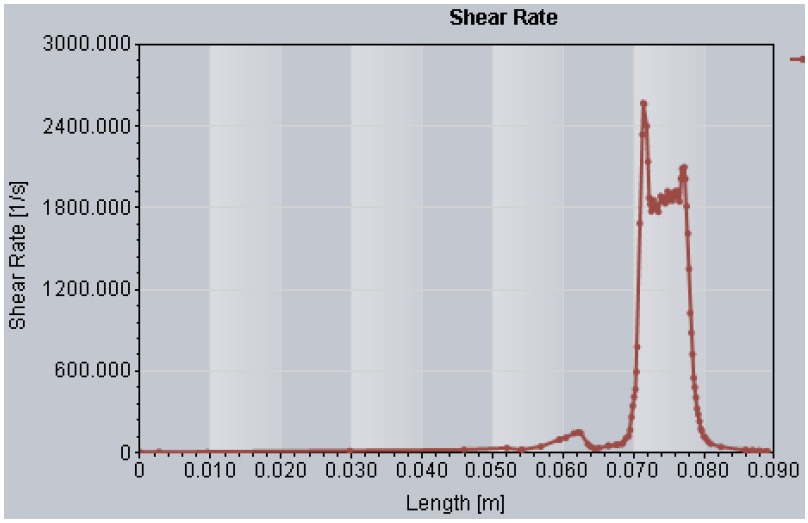
Length [m]

D.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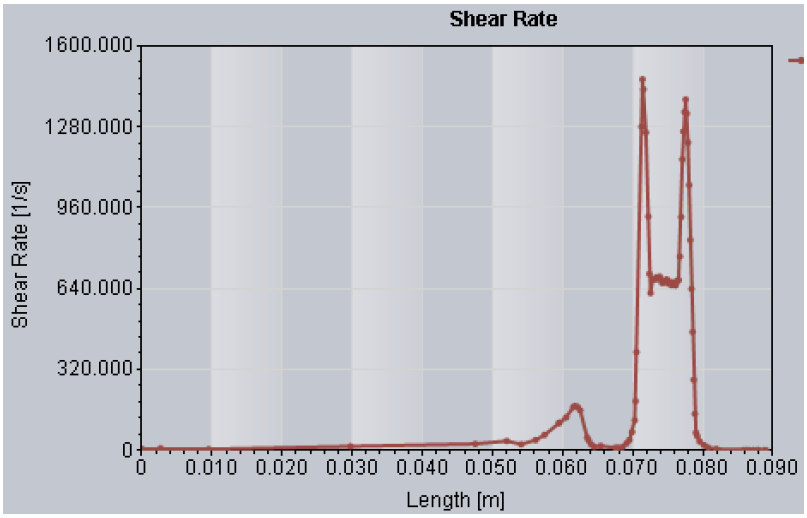
E. 

Figure 4: *Shear Rate Plots along the length of the stenosed ICA, where the throat of stenosis occurs at about Length=0.07m.*

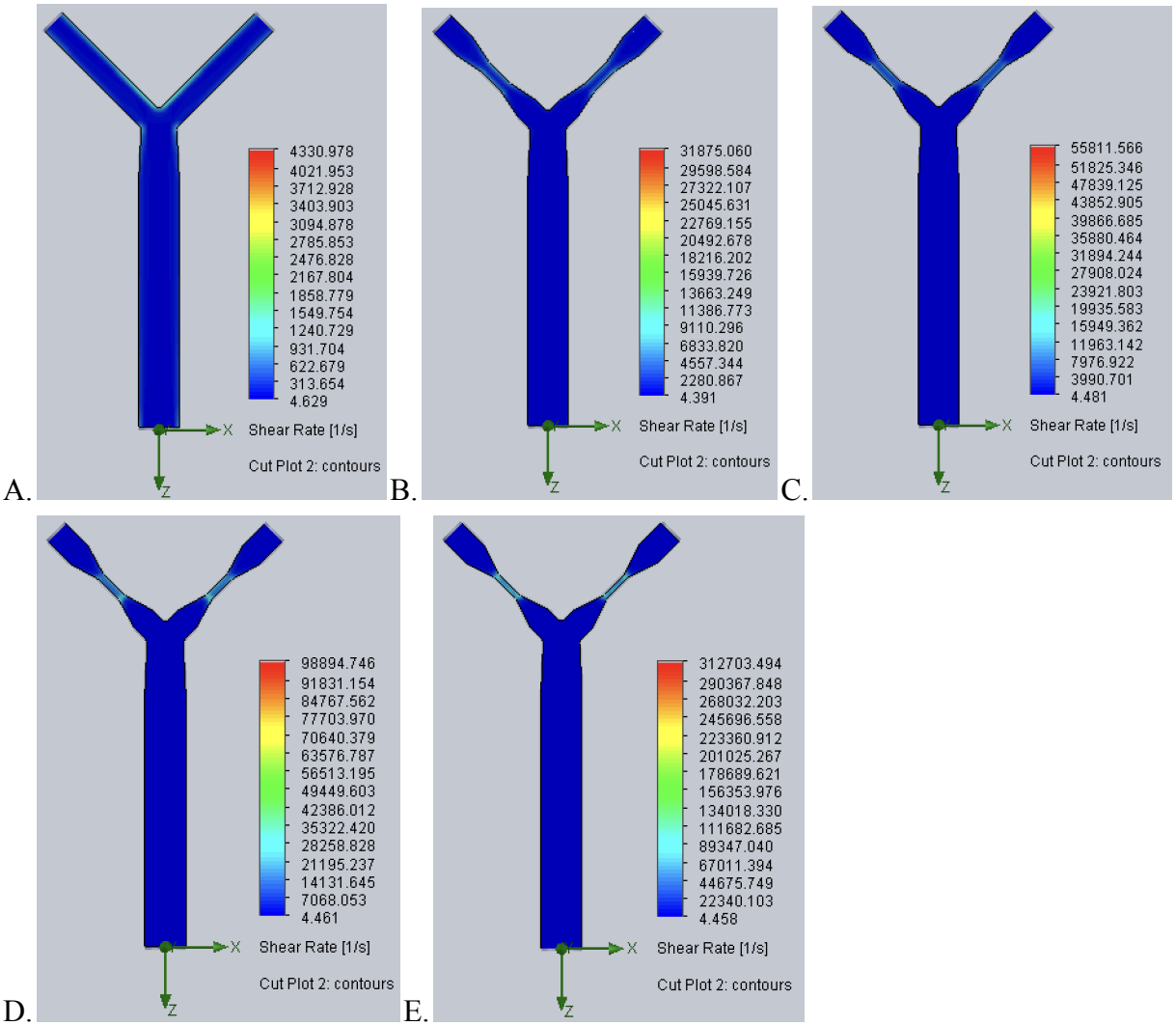


Figure 5: *Shear rate cut plots showing the changing shear rate at the throat of stenosis in the ICA and the ECA. Note: Shear rate correlates to shear stress at the same points of interest.*