

**PREDICTION OF CARDIOVASCULAR RISK: 2-D SIMULATION OF
ATHEROSCLEROTIC PLAQUE GROWTH IN IDEALISED HUMAN CAROTID
ARTERY**

AMOO TEMILOLUWA EMMANUEL

(13CF015124)

SEPTEMBER, 2021

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ARTERY**

BY

AMOO, TEMILOLUWA EMMANUEL

(13CF015124)

B.Eng Chemical Engineering, Covenant University, Ota.

**A DISSERTATION TO THE SCHOOL OF POSTGRADUATE STUDIES IN PARTIAL
FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF MASTER OF
ENGINEERING (M.Eng) DEGREE IN CHEMICAL ENGINEERING IN THE
DEPARTMENT OF CHEMICAL ENGINEERING, COVENANT UNIVERSITY, OTA,
NIGERIA.**

SEPTEMBER, 2021.

ACCEPTANCE

This is to attest that this dissertation is accepted in partial fulfilment of the requirement for the award of the degree of Master of Engineering (M. Eng.) in Chemical Engineering in the Department of Chemical Engineering, College of Engineering, Covenant University, Ota, Nigeria and has been accepted by the School of Postgraduate Studies, Covenant University, Ota, Ogun State.

Mr. John A. Philip

(Secretary, School of Postgraduate Studies)

.....

Signature and Date

Prof. Akan B. Williams

(Dean, School of Postgraduate Studies)

.....

Signature and Date

DECLARATION

I, AMOO TEMILOLUWA EMMANUEL (13CF015124) declare that this research was carried out by me under the supervision of Dr. Edith Alagbe of the Department of Chemical Engineering, College of Engineering, Covenant University, Ota, Nigeria. I attest that the dissertation has not been presented wholly or partially for the award of degree elsewhere. All sources of data and scholarly information used in this dissertation are duly acknowledged.

AMOO TEMILOLUWA EMMANUEL

.....

Signature and Date

CERTIFICATION

We certify that this dissertation titled “**PREDICTION OF CARDIOVASCULAR RISK: 2-D SIMULATION OF ATHEROSCLEROTIC PLAQUE GROWTH IN IDEALISED HUMAN CAROTID ARTERY**” is an original research work carried out by **AMOO, TEMILOLUWA EMMANUEL (13CF015124)** in the Department of Chemical Engineering, College of Engineering, Covenant University, Ota, Ogun State, Nigeria under the supervision of Dr, Edith Alagbe. We have examined and found this work acceptable as part of the requirements for the award of Master of Engineering in Chemical Engineering.

Dr. Edith E. Alagbe

.....

(Supervisor)

Signature and Date

Prof. Vincent E. Efeovbokhan

.....

(Head of Department)

Signature and Date

Prof. Oladipupo O. Ogunleye

.....

(External Examiner)

Signature and Date

Prof. Akan B. Williams

.....

(Dean, School of Postgraduate Studies)

Signature and Date

DEDICATION

This work is dedicated to the Almighty God, who gave me the grace to be able to embark on this project and finish well.

ACKNOWLEDGEMENTS

The Chancellor of Covenant University, Dr. David Oyedepo: a spiritual father and revolutionary leader whom God has used to mentor me. I would also like to acknowledge the entire management of Covenant University, starting from the Vice Chancellor, Prof. Abiodun H. Adebayo and the Dean, School of Postgraduate Studies, Prof. Akan B. Williams for ensuring a research-driven institution. Also, the Dean of Engineering, Prof. David O. Omole and the Head of Chemical Engineering Department, Prof. Vincent E. Efeovbokhan for their support.

Secondly, I would like to acknowledge the impact of my dear supervisor, Dr. Edith Alagbe, for her mentorship and guidance. She provided the full attention, moral and intellectual support needed for the success of this project. She got the data that was implement for my work for free. She also taught me perseverance and how to look deeper within myself and understand myself better.

I would like to acknowledge the impact of my friends; Oyedele Kayode from the Technical University of Munich, Germany, for his intellectual guidance and encouragement, and Dr. Paritosh Vasava of Arbonaut Ltd, Finland, for his mentorship. I also recognize the support of my course mates Neza Ezekiel, Abinusawa Adedoyin, Ekanem Godswill and Oboroghene Olomukoro

Lastly, I would like to acknowledge my dear parents Dr. and Dr. (Mrs) Amoo, for their unwavering love and sacrifice, their effort to ensure I have the best of life. Also, I appreciate my siblings Taiwo, Kehinde and Jumoke Amoo, for their support. To God be all the glory.

TABLE OF CONTENT

CONTENT	PAGE
ACCEPTANCE	i
DECLARATION	ii
CERTIFICATION	iii
DEDICATION	iv
ACKNOWLEDGEMENT	v
TABLE OF CONTENT	vi
LIST OF FIGURES	viii
LIST OF TABLES	x
LIST OF ABBREVIATIONS	xi
ABSTRACT	xii
CHAPTER ONE: INTRODUCTION	1
1.1 Background of the Study	1
1.2 Statement of the Problem	2
1.3 Aim and Objectives of the Study	3
1.4 Justification of the Study	3
1.5 Scope of the study	3
CHAPTER TWO: LITERATURE REVIEW	4
2.1 Atherosclerosis	4
2.1.1 Pathophysiology of Atherosclerosis	6
2.1.2 Risk Factors	8
2.1.3 Forms of Atherosclerosis	10
2.1.4 Treatment of Atherosclerosis	11
2.2 Newtonian and Non-Newtonian Fluid Characteristics	11
2.2.1 Newtonian Relation	12
2.2.2 Non-Newtonian Relation	13
2.3 Continuum Equations and Solution	18
2.4 Flow Modelling in Porous Medium	19
2.4.1 Capillary Bundle Concept	20
2.4.2 Darcy's Law	21
2.4.3 Biological Tissues as Porous Media	21

2.4.4	Mass Transport and Growth in Tissues	23
2.4.5	Rate of Fluid Flow and Solute Transport across Porous Media	25
2.4.6	Diffusion in tissues	27
2.5	Blood Characteristics and Modelling in Microvessels	28
2.6	Review of Past Works	30
CHAPTER THREE: METHODOLOGY		41
3.1	Blood Parameters	41
3.2	Boundary Conditions	41
3.3	Common Carotid Artery Geometry and Flow Parameters	43
3.4	Numerical Method	47
3.5	Modelling and Computational Set-Up	48
3.6	Mesh Independent Study and Validation of Result	49
3.7	Plaque Modelling and Growth Progression	50
3.8	Simulation Steps	50
CHAPTER FOUR: RESULT AND DISCUSSION		52
4.1	Results	52
4.2	Discussion of the Results	61
4.2.1	Initiation of Plaque Associated with Inlet Velocity	61
4.2.2	Variation of Plaque Height over Time	62
4.2.3	Effect of Stenosis Severity on WSS and PWS Distribution, Implication for Plaque Growth and Stroke Risk	63
4.2.4	Effect of Inlet Velocity on WSS And PWS Distribution, Implication for Plaque Growth and Stroke Risk	64
4.2.5	Effect of Blood Pressure Variation on WSS and PWS	65
4.2.6	Plaque Initiation and Development Associated with Blood Pressure	66
4.2.7	Discussions on Factors Affecting Blood Velocity and Pressure, Implication for Stroke and Control Measures	67
CHAPTER FIVE: CONCLUSION AND RECOMMENDTION		70
5.1	Conclusion	70
5.2	Contribution to Knowledge	71
5.3	Recommendation	70
REFERENCE		72
APPENDIX		87

LIST OF FIGURES

FIGURE	PAGE
2.1: Developmental overview of Atherosclerotic plaque	5
2.2: The pathophysiology of atherosclerosis	7
2.3: High-risk coronary plaque and significant stenosis on CT	10
2.4: Shear stress and rate data for Newtonian fluids	12
2.5: Rheogram showing Non-Newtonian fluid behavior	14
2.6: Tissue schematic diagram	22
2.7: Solute transport mechanism	23
2.8: Indication of how the Kedem-Katchalsky process of solute permeability	26
2.9: Non-Linear blood behaviour at difference heart cycles	29
3.1: Idealized carotid artery geometry constructed in COMSOL Multiphysics software	44
3.2: Inlet boundary where the inlet velocity boundary condition is applied	45
3.3: Outlet boundary where the outlet pressure boundary condition is applied	45
3.4: Lumen domain (highlighted in blue) where blood flows	45
3.5: Arterial wall domain (highlighted in blue) Linear elastic in property	46
3.6: Arterial wall interface with the lumen marking blood flow boundary	46
3.7: External arterial wall characterized as free boundary (highlighted in blue)	46
3.8: Constrained arterial edge (highlighted in blue for model stability)	47
3.9: Meshing of the Arterial domains (triangular meshing)	49
4.1: Plaque growth at the initiation stage variation with inlet blood velocity (after 304 days)	52
4.2: Differential increase in plaque height every 304 days for 5 years	52
4.3: Plaque height every 304 days for 5 years	53
4.4: WSS distribution along the arterial length for 30 cm/s inlet velocity	53
4. 5: PWS distribution along the arterial length for 30 cm/s inlet velocity	54

4.6: Velocity contour plot for no plaque state of the arterial for 30 cm/s inlet velocity	54
4.7: Velocity contour plot for initiation stage (17% stenosis) for 30 cm/s inlet velocity	55
4.8: Velocity contour plot for 50 stenosis stage for 30 cm/s inlet velocity	55
4.9: Velocity contour plot for 50 stenosis stage for 30 cm/s inlet velocity	56
4.10: WSS distribution along the arterial length at 80% degree of stenosis	56
4.11: PWS distribution along the arterial length at 80% degree of stenosis	57
4.12: WSS distribution along the arterial length at initiation stage (17.3 %) at different outlet pressure at 30 cm/s inlet velocity	57
4.13: PWS distribution along the arterial length at initiation stage (17.3 %) at different outlet pressure	58
4.14: WSS distribution along the arterial length at 50% stenosis stage at different outlet pressure at 30 cm/s inlet velocity	58
4.15: PWS distribution along the arterial length at 50 % stenosis stage at different outlet pressure.	59
4.16: WSS distribution along the arterial length at 80% stenosis stage at different outlet pressure at 30 cm/s inlet velocity	59
4.17: PWS distribution along the arterial length at 80 % stenosis stage at different outlet pressure	60
4.18: Differential increase in plaque height every 304 days for 5 years.	60
4.19: Overall plaque height for 5 years	61

LIST OF TABLES

TABLE	PAGE
2.1: Hypertension Definition	8
2.2: The cell growth kinetic models	24
3.1: Carotid artery dimension and properties	41
3.2: Inlet velocity of test subjects	43
3.3: Carotid artery dimension and properties	44

LIST OF ABBREVIATIONS

ABBREVIATION	DESIGNATION
WSS:	Wall Shear stress
WTI:	Wall Thickness Increase
PWS:	Plaque Wall Stress
CVD:	Cardiovascular Disease
CHD:	Coronary Heart Disease
BP:	Blood Pressure
LDL:	Low Density Lipoprotein
HDL:	High Density Lipoprotein
oxLDL:	Oxidized Low Density Lipoprotein
oxHDL:	Oxidized High Density Lipoprotein
FSI:	Fluid Solid Interaction
CFD:	Computational Fluid Dynamics

ABSTRACT

Atherosclerosis is one of the leading causes of death worldwide. There is a dearth of data and complexity in scientific research to predict the severity of the disease, the period of crisis and hemodynamic conditions that can lead to a stroke. This work focused on plaque growth in a 2-D carotid artery model that is dependent on the hemodynamics: wall shear stress (WSS), and the mechanical plaque response: plaque wall stress (PWS). A linear two-dependent-variable plaque growth model was used to simulate the rate of initiation and progression of the plaque overtime. The COMSOL Multiphysics v5.5 software was used to solve the fluid / solid dynamics problems where PWS and WSS values were extracted. The WSS and PWS showed a strong dependence on blood velocity and pressure. Increased blood velocity was associated with increased WSS and PWS, this resultantly led to a decrease in plaque growth rate. Maximum stenosis degree was 30 % after 5 years of exposure of 30 cm/s average blood inlet velocity, whereas the minimum of 22 % was obtained for average blood inlet velocity of 45 cm/s. The effect of increased blood pressure was more eminent at the root and regions on the arterial wall distal from the plaque neck, where increased blood pressure caused an increased WSS and PWS. Maximum stenosis degree was 32 % after 5 years of exposure of 1500 Pa average outlet pressure, whereas minimum stenosis of 29.5 % was obtained for average outlet pressure of 0 Pa for the same period. Variations in blood velocity had a more significant effect on plaque growth in relation to variations in blood pressure. Plaque progression had an inverse relationship with blood velocity, however a direct relationship of plaque growth with pressure was found. Higher plaque heights showed dangerously high PWS and WSS that could cause a potential rupture of plaque around regions localized at the root of the plaque. At stenosis degrees from 50 % and above, maximum WSS and PWS obtained were 580 Dyne /cm² and 33 kPa respectively, of which the WSS value shot above the threshold of 112 Dyne/cm², hence indicating plaque rupture risks. People that experience higher blood pressure and low velocity were at higher risk of atherosclerosis progression and ultimately a stroke crisis. It would be recommended that more patient-specific studies based on these models should be considered and studied in future.