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RESPONSE INTERACTION OF ARTERY UNDER MECHANICAL VIBRATION

S. I. KUYE[1], S. O. ISMAILA[1], O. S. OLOKODE[1] AND I. O. ABIALA[2]

[1]Department of Mechanical Engineering, College of Engineering, Federal University of Agriculture, Abeokuta. Postcode 110001. Nigeria
[2]Department of Mathematics, College of Natural Sciences, Federal University of Agriculture, Abeokuta, Nigeria
*__Corresponding author__: ibiyemikuye@yahoo.com **Tel:** +2348033018030

ABSTRACT

Hypertension or high blood pressure, sometimes called arterial hypertension, is a chronic medical condition in which the blood pressure in the arteries is elevated. The aim of this work was to analyse mathematically the vibration of the artery and show its relationship to hypertension related cases. Principles of conservation of mass and momentum inline with Euler-Bernoulis method were used. By idealising the artery resting on the tissue as an elastic beam that is resting partially on an elastic continuum and simply supported, a boundary value partial differential equation governing the blood- artery - interaction mechanics was formulated. The governing partial differential equation for the problem was obtained and after linearization was solved using integral methods of Fourier-Laplace Transform. Simulations for some cases were carried out varying some choice parameters. Findings show that natural frequency of the artery decreases with increase in the mean arterial pressure (MAP). It was equally found out that vibration of the artery generally increases with increase in MAP and increase in mode. It was thus concluded that mechanical vibration of the artery increases with increase in blood pressure, and continuous vibration can lead to fatigue and failure which can cause obstruction in the supply of blood to other parts of the body and may lead to stroke.

Keywords: Artery, hypertension, deformation, vibration.

INTRODUCTION

According to the general stress concept, repeated temporal changes in biological responses can result in permanent metabolic changes of the organism leading to chronic diseases in the long run (Barbisch, 2011).

Man, as a mechanical system, is extremely complex and his mechanical properties readily undergo change. There is limited reliable information on the magnitude of the forces required to produce mechanical damage to the human body (Gierke & Brammer, 2002). Dynamic arterial blood pressure and blood flow are key determinants of normal or pathological functioning of the cardiovascular system (Rose *et al.*, 2008). If a structure is sufficiently flexible, the structural deformation under the fluid loading will in turn change the fluid force. The response can be unstable with very large structural vibrations—once the fluid velocity exceeds a critical threshold value (Blevins, 2002). Mookerjee *et al.* (2008) used mathematical model to simulate the pressure prop-

agation characteristics in large elastic arteries defined by some input parameters. The pressure wave characteristics were then analysed to estimate carotid-femoral pulse wave velocity. They indicated that their predictions closely match clinically observed trends. Rose *et al*. (2008) developed a computational model of systemic arterial hemodynamics. The model predicts dynamic pressures and flows throughout the systemic arterial vascular bed. Hyre and Pulliam (2008) developed a computational model capable of predicting balloon/stent/artery/plaque interactions and their effects on arterial and plaque stresses to assess the effects of length mismatch and plaque classification on stent expansion characteristics and arterial stresses in diseased arteries. In most previous studies, the arterial wall was assumed to behave as a rigid wall without deformation or motion. However, the exact nature of the problem lies in the two way interactions between dominant pulsatile flow and the deformable arterial wall structure (Rose & Johnson, 2008). Hypertension or high blood pressure, sometimes called arterial hypertension, is a chronic medical condition in which the blood pressure in the arteries is elevated. This requires the heart to work harder than normal to circulate blood through the blood vessels (Wikipedia, 2012). It may result in a stroke that is a medical emergency and which in turn can cause permanent neurological damage, complications and death (Donnan *et al*., 2008, Wikipedia, 2012). Stroke is the second leading cause of death worldwide (Mathers *et al*., Wikipedia, 2012) This work was intended to show the link between high blood pressure and mechanical vibration of the artery.

A series of experiments by Aitken (1878), on traveling chains and elastic cords, illustrating the balance between motion-induced tensile and centrifugal forces, is perhaps among the earliest work pertinent to the study of dynamics of flexible pipes conveying fluid. Similarly, the first observation of the peculiar spontaneous motions imparted to the free end of a rubber pipe, such as might be used to water the lawn, by a sufficiently high flow rate was made long ago, evidently, this was first recognized as a self-excited oscillation by Marcel Brillouin in 1885 as reported by Bourrieres (1939), one of Brillouin's students. Bourrieres (1939) was the first to undertake a serious study of the dynamics of flexible pipes conveying fluid. In his work, he examined the oscillatory instability of cantilevered pipes conveying fluid, both theoretically and experimentally. He derived the correct equation of motion and, although he was unable to obtain analytically the critical flow velocity for the onset of the oscillation, he nonetheless determined most of the salient characteristics of the phenomenon. Paidoussis found that vertical, continuously flexible pipes are never subject to buckling. This was clarified by Paidoussis and Deksnis (1970). Semler *et al*. (1994) derived a complete set of geometrically nonlinear equations of motion of fluid conveying pipes. They accounted for large strains and assumed the kinematics of the Euler-Bernoulli beam theory. They used both the energy and Newtonian approaches to derive the equations of motion and showed them to be identical. Gorman *et al*. (2000) more recently formulated a generalized model which includes the effects of radial shell vibration of pipes and initial axial tensions within the pipes besides both the Poisson and friction coupling mechanisms and concluded that transient hydrodynamic pressure waves trigger a vibration of the pipeline with a higher frequency and that with increasing pulsation frequencies, longitudinal vibration tends to

be larger. In their book, Mushtari and Galimov (1957) presented non-linear theories for moderate and large deformations of thin elastic shells. The non-linear theory of shallow shells is also discussed in the book of Vorovich (1999). Olunloyo *et al*. (2005, 2007) studied the pipe walking phenomenon in addition to the transverse and longitudinal vibrations alongside the effect of elevated temperature and concluded that the role of the transient solution may not be as central for pipe walking as was hitherto believed as there are other significant contributions emanating from some of the other parameters.

The human cardiovascular system is a complex hydraulic network coupled to an oscillatory pump. Abnormal values of pressures and flows in the system can be signs, symptoms, and causes of pathology (Rose *et al*., 2008).

An important goal of this study was to analyse the dynamic interaction of artery under mechanical vibration induced by the fluid forces imposed due to blood conveyance.
To accomplish this goal, principles of conservation of mass and momentum were used to derive the partial differential equations governing the dynamics of the artery subjected to change in blood pressure in line with Euler-Bernoullis method. The linearised equations were solved using Fourier-Laplace Transform. Simulations were carried out for a particular case.

Arterial pressure analysis

The next few outlines describe the different form of blood pressure measurements that can be obtained within the human body system.

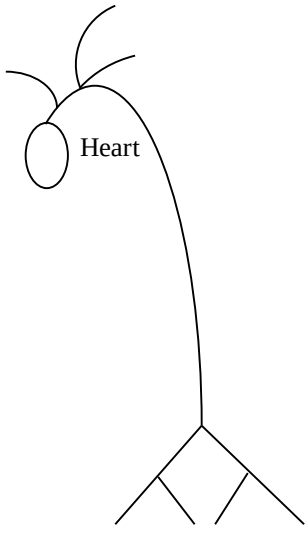

Figure1: Systemic Vascular Arterial Network

Systolic Pressure

Systolic pressure is the maximum pressure exerted by the blood against the artery walls. It is the result of ventricular systole or contraction. It is normally about 120 mmHg.

Diastolic Pressure

Diastolic pressure is the lowest pressure in the artery. It's a result of ventricular diastole (relaxation) and is usually around 80 mm Hg.

Pulse Pressure

Pulse Pressure is the difference between systolic and diastolic pressure. It is the throb you feel when you take your pulse.

Pulse Pressure = Systolic Pressure - Diastolic Pressure

~ 40 mm Hg ~120 mm Hg - ~80 mm Hg

Mean Arterial Pressure

Mean Arterial Pressure (MAP) is a calculated "average" pressure in the arteries.

Mean Arterial Pressure (MAP) = Diastolic Pressure + 1/3 Pulse Pressure

~93 mm Hg = ~80 mm Hg + ~ 40/3 mm Hg

MAP is closer to the diastolic pressure than systolic pressure because the heart stays longer in diastole.

MAP is the force that propels the blood through the arteries. Since the MAP is the force that propels the blood through the arteries, it implies that the MAP has to remain within optimum range (about 93mm Hg) to prevent rupture of artery in any form of blood clots (Cummings, 2012).

PROBLEM FUNDAMENTALS AND GOVERNING DIFFERENTIAL EQUATION

The physical problem under investigation consists of a pre-stressed artery conveying pressurized blood from the heart and is subjected to vibration as a result of the load being carried. The artery considered is assumed to be simply supported but the supports are buried in the surrounding tissues. The underlying theory employs the following hypotheses namely:

(i) The pre-stressed artery is idealized as an elastic beam while the surrounding tissue is modeled as a Winkler foundation.

(ii) A fully developed incompressible pressurized blood is flowing through the portion considered.

(iii) The blood flow is assumed Newtonian.

(iv) The system is under the influence of hydrodynamic, bending loads, and internal fluid flow forces.

(v) The pre-stressed blood conveying artery is subjected to infinitesimal strains.

The elastically deforming artery is subjected to Gorman *et al*. (2000) infinitesimal strains.

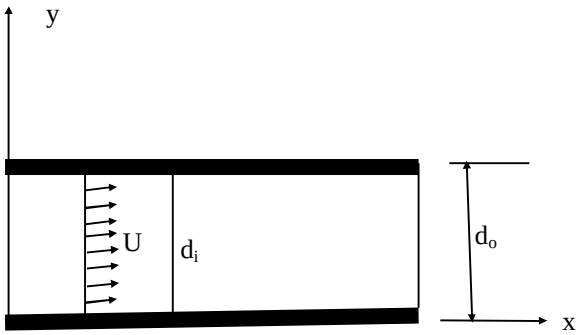

Figure 2: A Model of Blood-Artery System Before Deformation

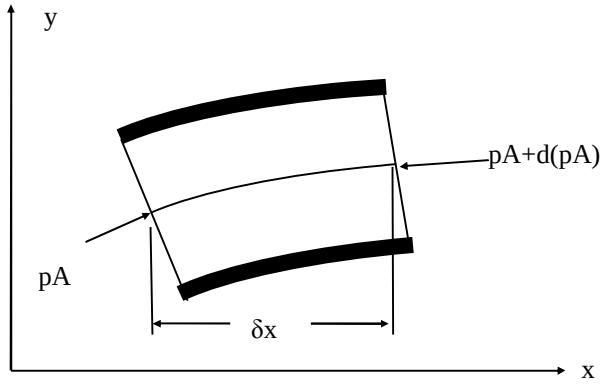

Figure 3: A Model of Blood-Artery System After Deformation

ANALYSIS OF THE VIBRATION

Following the foregoing assumptions, the linearised formulated governing differential equation for the artery conveying blood is given below as

$$\left\{ EI\frac{\partial^4 w}{\partial x^4} + M\frac{\partial^2 w}{\partial t^2} + 3m_f U^2 \frac{\partial^2 w}{\partial x^2} - (To - pA)\frac{\partial^2 w}{\partial x^2} \right\} + (pA)' \frac{\partial w}{\partial x} + c\frac{\partial w}{\partial t} + c_D \frac{\partial w}{\partial t} + k_b w = F_o e^{j\Omega t} \quad (1)$$

where

To simplify Eq. (1), we invoke Olunloyo *et al.* (2007) procedural analysis namely:

$$A = A_o\left(1 - \gamma\frac{x}{L}\right), A' = -A_o\frac{\gamma}{L}$$

$$\left[(pA)' = \left(\frac{-\Delta p}{L}\right)A_o + p_o A'\right]$$

$$= \frac{-p_o A_o \gamma}{L} - \frac{\Delta p}{L}A_o(1-\gamma)$$

$$and\ A' = -A_o\frac{\gamma}{L} \quad (2)$$

This allows us to rewrite Eq. (1) as

U = Velocity of blood flow

$$\left\{\begin{array}{l} EI\dfrac{\partial^4 w}{\partial x^4}+M\dfrac{\partial^2 w}{\partial t^2}+\left(\begin{array}{l}3m_bU^2-T_o\\+pA_o(1-\gamma)\end{array}\right)\dfrac{\partial^2 w}{\partial w^2} \\ -\dfrac{p_oA_o\gamma}{UL}\dfrac{\partial w}{\partial t}-\dfrac{\Delta p}{UL}A_o(1-\gamma)\dfrac{\partial w}{\partial t}+c_D\dfrac{\partial w}{\partial t} \\ +k_bw=F_oe^{i\Omega t} \end{array}\right\} \quad (3)$$

Introducing Laplace Transform to Eq. (3) leads to

$$\left\{\begin{array}{l} EI\dfrac{d^4 w}{dx^4}+M\left(s^2\widetilde{w}(x,s)+s\widetilde{w}(x,0)+\widetilde{w}(x,0)\right) \\ +\left(3m_bU^2-T_o+pA_o(1-\gamma)\right)\dfrac{d^2\widetilde{w}}{dx^2} \\ -\left(\dfrac{p_oA_o\gamma}{UL}+\dfrac{\Delta p}{UL}A_o(1-\gamma)-c_D\right)\left(\begin{array}{l}s\widetilde{w}(x,s)\\-\widetilde{w}(x,0)\end{array}\right) \\ +k_b\widetilde{w}=F_oe^{i\Omega t} \end{array}\right\} \quad (4)$$

Using zero initial conditions $\widetilde{w}(x,0)=\widetilde{\dot{w}}(x,0)=0$ enables Eq. (4) to be written as

$$\left\{\begin{array}{l} EI\dfrac{d^4\widetilde{w}}{dx^4}+Ms^2\widetilde{w}+\left(\begin{array}{l}3m_bU^2-T_o\\+pA_o(1-\gamma)\end{array}\right)\dfrac{d^2\widetilde{w}}{dx^2} \\ +\left(c_D-\dfrac{p_oA_o\gamma}{UL}-\dfrac{\Delta p}{UL}A_o(1-\gamma)\right)s\widetilde{w} \\ +k_b\widetilde{w}=\dfrac{F_o}{s-i\Omega} \end{array}\right\} \quad (5)$$

Introducing Fourier transformation to Eq. (5) and applying boundary conditions for simply supported beam gives

$$\widetilde{w}^F(\lambda_n,s)=\left\{\frac{F_o1^F}{M(s-i\Omega)(s^2+l_1s+l^2)}\right\} \quad (6)$$

where,

$$l_1=\frac{\left(c_D-\dfrac{p_oA_o\gamma}{UL}-\dfrac{\Delta p}{UL}A_o(1-\gamma)\right)}{M} \quad (7) \text{ and}$$

$$l^2 = \frac{EIn^4\pi^4 - \left(3m_bU^2 - T_o + pA_o(1-\gamma)\right)n^2\pi^2 + k_b}{M} \tag{8}$$

On invoking Fourier-Laplace inversion, solution to Eq. (6) becomes

$$w(x,t) = \frac{2}{L}\sum_{n=1}^{\infty}\frac{F_o\Gamma(t)}{M}\frac{L\left(1+(-1)^{n+1}\right)}{n\pi}\sin\frac{n\pi x}{L} \tag{9}$$

where,

$$\Gamma(t) = \left\{\begin{array}{l} \dfrac{e^{i\Omega t}}{(\alpha_1+i\Omega)(\alpha_2+i\Omega)} + \dfrac{e^{-\alpha_1 t}}{(i\Omega-\alpha_1)(\alpha_2-\alpha_1)} \\ -\dfrac{e^{-\alpha_2 t}}{(i\Omega-\alpha_2)(\alpha_2-\alpha_1)} \end{array}\right\} \tag{10}$$

and

$$\alpha_1 = \frac{\ell_1}{2} + i\sqrt{\ell^2 - \frac{\ell_1^{\,2}}{4}}$$

$$\alpha_2 = \frac{\ell_1}{2} - i\sqrt{\ell^2 - \frac{\ell_1^{\,2}}{4}} \tag{11}$$

RESULTS

Table 1: Table of parameters used

S/N	DESCRIPTION	SYMBOL	VALUES USED
1.	Mean Artery Pressure	p_1	93 mmHg (12,408 N/m2)
2.	Difference in Pressure	Δp	2,408 N/m2
3.	Artery Internal Diameter	d_1	2.85 mm
4.	Artery thickness	T	0.3 mm
5.	Pre-stressed Tension	T_o	15 N/m2
6.	Drag Coefficient	c_D	1
7.	Damping Force per Unit Velocity	c	0.2
8.	Stiffness of the Artery	k_b	0.1
9.	Artery's Modulus of Elasticity	E	5 x 105 N/m2
10.	Moment of Inertia	I	1.98 x 10-12 m4
11.	Density of Artery	ρ	900 kg/m3
12.	Density of Blood	ρ_f	1050 kg/m3
13.	Length of Artery Considered	L	5 cm

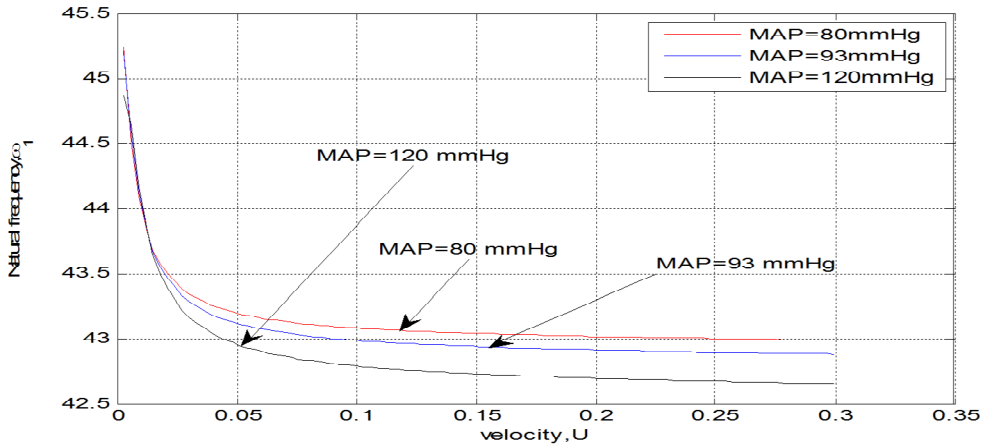

Figure 4: Plot of Primary Natural Frequency ω_1 versus Velocity for Varying MAP Values

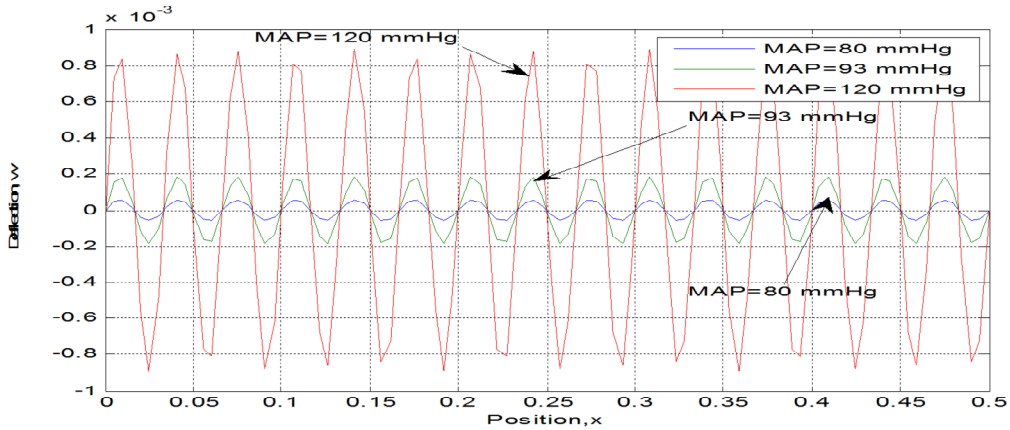

Figure 5: Plot of Response w versus Position for Varying MAP

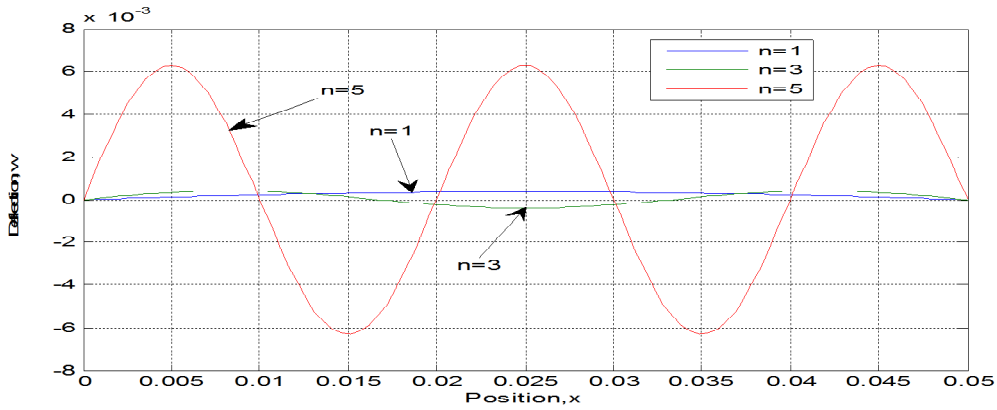

Figure 6: Plot of Response w versus Position for Varying Mode for MAP=93 mmHg

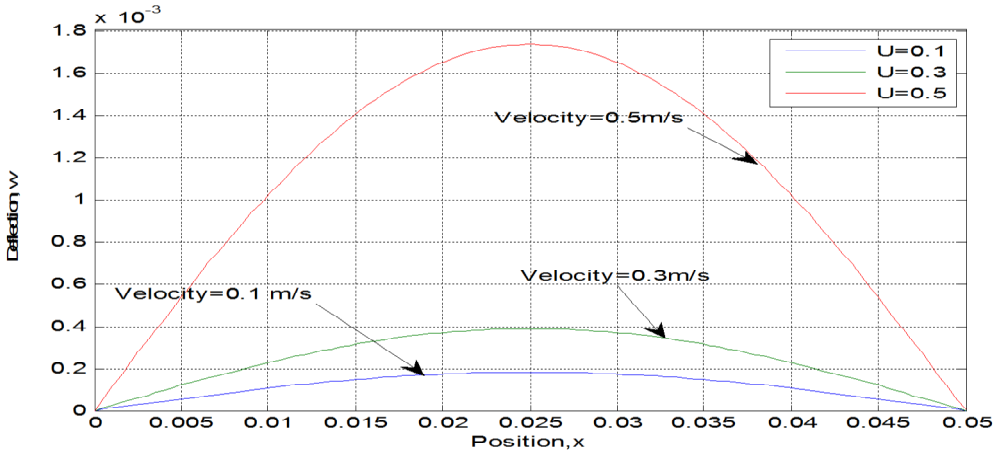

Figure 7: Plot of Response w versus Position for Varying Velocity for MAP=93 mmHg

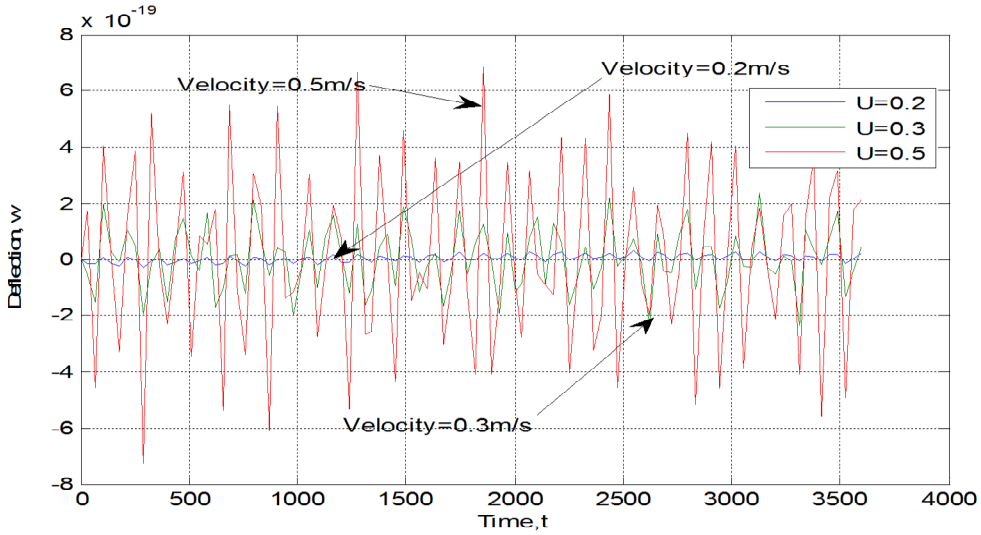

Figure 8: Plot of Response w versus Time for Varying Velocity for MAP=93 mmHg

RESULTS AND DISCUSSION

The response of the artery under heart-induced mechanical vibration is considered in this work. Figure 4 is a graph of primary natural frequency against velocity of blood low for different mean arterial pressures (MAP). It could be seen that the natural frequency started from the maximum to a minimum where it became constant with increase in velocity of flow. Generally, the graph shows that natural frequency of the artery considered decreases with increase in MAP i.e. in the order 120 mmHg > 93 mmHg > 80 mmHg. Decrease in the natural frequency of an object exposes it to vibration when there is excitation. Figure 5 is the graph of complimentary natural frequency against velocity of blood flow. It equally shows that complimentary natural frequency decreases with increase in MAP. When the

excitation frequency gets near one of the natural frequencies, resonance is set up, this causes serious vibration of the artery that may lead to more problems such as fatigue, situation called walking and even failure if care is not taken. Figure 6 shows the graph of Response against Position of the artery at different MAP, it can be observed that response increases with MAP in the order 120 mmHg > 93 mmHg > 80 mmHg, i.e. the more the MAP the more the deformation being experienced. The artery vibrates more as the blood pressure increases. When the blood pressure increases to a particular level unchecked, failure may occur in the artery. This generally agrees with literature for pipes conveying pressurized fluid. The graph of Response against Position of the artery for different modes is shown by Figure 6 at MAP 93 mmHg. It can be seen that deflection increases with modes in the order n=5>n=3>n=1.

Figure 7 is a graph of Response against Position for varying velocity. Deflection also increases with velocity. The higher the velocity of blood flow the more the vibration and the more the risk of failure. Graph of Response against Time is depicted by Figure 8. Deflection increases in this case also with increase in velocity. This is similar to the deduction in Figure 7.

When vibration sets in unchecked, the artery is subjected to stress, the surrounding tissues experience fatigue and the body generally is affected. This is likely why headache is among the signs of high blood pressure.

CONCLUSION

The goal of this study was to analyse the dynamic interaction of artery under mechanical vibration. To accomplish it, differential equation governing the problem was formulated and solved using Fourier-Laplace method.

The results obtained were in agreements with literatures on vessels conveying pressurized fluid. This work will throw more light on how increase in blood pressure leads to failure of blood conveying vessels in the body.

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