

# Mathematical Analysis of Pulsatile Flow of Blood in Bifurcation with Varying Frequency

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**Abstract** - Mathematical Model for carotid artery bifurcation with flow direction ratio ( I : E ) at the apex is studied in the present paper. Study consists the blood flow in Y model with flow rate ( I : E ) = 70% : 30% between internal carotid artery (ICA) and external carotid artery (ECA) respectively from the neck of the common carotid artery (CCA). The behavior of periodic dependent pulsatile flow, a representative normal carotid artery bifurcation wave form is imposed at the common carotid artery (CCA). To produce a representative constant flow split between ICA and ECA, fully developed velocity profile is applied in CCA. Peak systolic and end diastolic pressures are compared with cardiac cycle. The flow under consideration is associated with the difference between Reynold's number at a different radius of the artery leads to the flow stability breaks and forms the vortex at the junction of bifurcation. This is most possible case when there appears sudden change in the artery diameter. This behavior is numerically studied using Strouhal's number which is given by  $St = f \times R \bar{V}$ . Pressure at the apex and velocity profiles under steady pulsatile wave form have been studied with frequency variations on each location in the direction of the flow. Peak systolic and diastolic pressures have been studied for 34 time steps. Series solution method is employed to study the governing equations of motion with perturbations on the characterizing physiological parameters.

**keywords** - Artery, pulsatile, flow, velocity, blood

## I. INTRODUCTION

The study of blood flow through bifurcations in the arterial system has been of special interest because of its connection with certain forms of arterial diseases. Studies have shown that atherosclerosis is much more likely to occur in the proximal of vessel branches and junctions. As the fluid passes through a bifurcation, its direction changes which results in flow separation, secondary flow and pressure shear stress variations along the wall. If the flow stability breaks down due to varying radius then there appears rapid time variant flow as directly related to the formation of vortex. Numerically it can be identified by Strouhal's Number. Many researchers have reported that the materials trapped in the stasis zone might deteriorate and cause degeneration of the wall. In addition, within the recirculation zone, oscillatory change of direction and magnitude of the wall shear stress could damage the endothelial cells, which form a protective lining within the vessel. The streamlines of the flow towards the wall due to instability the sinus ridge becomes stagnation point so that vortex formation is governed by the duration of the systolic, quantitatively described by Strouhal's number. The clinical study shows the lesions are localized more on the inner corner of the branch artery where the shear stress was taken 'high' in the case of femoral arteries. Idealization of the vascular system with respect geometry and structure makes it possible to describe quantitatively many phenomena that are of physiological interest. The cusps from being pushed into sinuses (to form the resulting flux) and the occlusion generates the orifices of coronary sinus. The cusps generated in the coronary sinus play an important role in the normal function of the flow of blood during systole in the coronary arteries.

The complex pathology of atherosclerosis appears as a disease of the inner layer of the arteries. It may begin on the interface between arterial wall (the endothelial surface) and blood or just within the intima, the layer of tissue closest to the blood interface. Therefore atherosclerosis is affected by those physical and chemical events, which cause and control the aggregation of blood platelets. The hemodynamic factors such as change of velocity, pressure and transmission of volume have a major role in the development of complex blood flow patterns in the carotid artery bifurcations. The common carotid artery (CCA) at the neck divides into the internal carotid artery (ICA) and external carotid artery (ECA). The ICA supplies the blood to the brain with and generally dilates as it's offspring (the carotid sinus or bulb). Because of atherosclerotic lesions that often occur proximally in the bulb, the ICA is an interesting subject to investigate into whether local flow velocity patterns can determine atherosclerosis.

Bharadvajet. al. [1] studied a model of human carotid bifurcation for steady flow through flow visualization. Porentaet. al. [2] analyzed a finite element model of blood flow in arteries which includes taper, branches and obstructions. Kaluzynskiet. al. [4] investigated the velocity distributions due to effect of wall roughness by considering a model of the carotid artery bifurcations using ultra sound LDA. Rene Botnaret. al. [5] studied a model to compare numerical simulations and in-vitro MRI measurements in carotid artery bifurcation. Basavarajappaet. al. [6] studied the experimental and numerical analysis of pulsatile flow in carotid artery bifurcations. JaehoonSeonget. al. [7] studied the morphological age-dependent development of the human carotid bifurcation. Mandru Met. al. [8] explained the mechanical properties of native and bio-mimetically formed arterial tissues using data from the energy function. T Kopplet. al. [9] studied the impact of varying

degrees of unilateral stenosis of a carotid artery on pulsatile blood flow and transport of oxygen from heart to the brain. Yakov A Gataulin et al. [10] discussed the study of flow in two geometrically different models of the common carotid artery, the statistically average one and the one with maximum physiological tortuosity and examined the temporal and spatial evolution of swirling blood flow depending on curvature parameters of the artery. S Balamuralitharan [11] investigated the bifurcation analysis of nonlinear system of SIR epidemic model with treatment. Isaac Mwangi Wangari [12] investigated the effect of recurrent TB on the formation of backward bifurcations through detailed mathematical modelling. Yakin Shu et al [13] proposed a mathematical model of interactions between tumor cells, M1 and M2 macrophages.

The present study concerns the analysis of pulsatile flow of blood with rapidly time variant flow. The flow under consideration is associated with the difference between Reynold's number at a different radius of the artery leads to the flow stability breaks and forms the vortex at the junction of Bifurcation. This is most possible case when there appears sudden change in the artery diameter. This behavior will be numerically studied using Strouhal's number  $S_t$ . Further, it is  $S_t$  related to Reynold's Number which also describes the role in pressure flow determination of the velocity which behaves as oscillatory flow.

**II. FORMULATION**

Since the carotid artery is distensible, we assume the tapering of the arterial wall radius as the axial distance increases away from the apex.  $R$  be the radius of CCA  $R_I$  be the radius of ICA and  $R_E$  be the radius of ECA and ( $R_E < R_I$ ), as shown in the fig (1). The ICA is tapered from 3.3 mm at the neck termed 0.0 mm (axial) in to the ICA to 2.4 mm at a point 10.0 mm in to the ICA. Analyzing the tapering, the radius is given by,

$$R = R_0 - Z \tan\theta \tag{1}$$

Where  $R_0$  – undisturbed radius,  $Z$ - location,  $\theta$  - angle of tapering.

Equation (1) is valid for rigid tube. For an elastic tube radius of ICA is,

$$R_I = R \left[ 1 \pm \varepsilon \sin \frac{2\pi}{\lambda} (z - ct) \right] \tag{2}$$

Where  $\varepsilon$  - amplitude ratio for the distensible wall,  $\lambda$  - wavelength,  $c$ - wave speed,  $t$  – time.

For irregular wave

$$R_I = \begin{cases} R_0 & , z < 0 \\ R_0 - \frac{\delta}{2} \left[ 1 + \cos \left( \frac{n(z - z_1)}{z_0} \right) \right] & , 0 < z < z_0 \\ R_0 & , z > 2z_0 \end{cases}$$

where  $z_0$  is the half length,  $\delta$  - thickness of the plaque.

In the tube the flow is pulsatile. The arterial motion is sinusoidal. This along with the pressure difference helps the blood to flow further in to the artery. Wall movements of bifurcation are shown in fig (1). The wavelength ( $\lambda$ ) in ICA and CCA is not same. Therefore in both the tubes the flow velocities will not be equal. Volumetric flow in ICA is 70% of flow in CCA. Navier-Stokes equations that describes the motion of the fluid in the model are taken radial and axial directions.

$$\rho \frac{\partial \bar{u}}{\partial t} = -\frac{\partial \bar{p}}{\partial r} + \mu \left[ \frac{\partial^2 \bar{u}}{\partial r^2} + \frac{1}{r} \frac{\partial \bar{u}}{\partial r} - \frac{\bar{u}}{r^2} + \frac{\partial^2 \bar{u}}{\partial z^2} \right] \tag{3}$$

$$\rho \frac{\partial \bar{v}}{\partial t} = -\frac{\partial \bar{p}}{\partial z} + \mu \left[ \frac{\partial^2 \bar{v}}{\partial r^2} + \frac{1}{r} \frac{\partial \bar{v}}{\partial r} + \frac{\partial^2 \bar{v}}{\partial z^2} \right] \tag{4}$$

With the continuity equation,

$$\frac{1}{r} \frac{\partial}{\partial r} [r \bar{u}] + \frac{\partial \bar{v}}{\partial z} = 0 \tag{5}$$

Taking dimensionless quantities

$$v = \frac{\bar{v}}{v_0}, u = \frac{\bar{u}}{u_0}, p = \frac{\bar{p}}{\rho v_0^2}, t = \frac{\bar{t} v_0}{R_0}, z = \frac{\bar{z}}{z_0}, r = \frac{\bar{r}}{R_0}, \text{with } z_0 \approx R_0 \tag{6}$$

Then equations (3.3) and (3.4) becomes,

$$\frac{\partial u}{\partial t} = -\frac{\partial p}{\partial r} + \frac{1}{R_e} \left[ \frac{\partial^2 u}{\partial r^2} + \frac{1}{r} \frac{\partial u}{\partial r} - \frac{u}{r^2} + \frac{\partial^2 u}{\partial z^2} \right] \tag{7}$$

$$\frac{\partial v}{\partial t} = -\frac{\partial p}{\partial z} + \frac{1}{R_e} \left[ \frac{\partial^2 v}{\partial r^2} + \frac{1}{r} \frac{\partial v}{\partial r} + \frac{\partial^2 v}{\partial z^2} \right] \tag{8}$$

where,

$$R_e = \left[ \frac{\mu}{\rho v_0 R_0} \right]^{-1},$$

$R_e$  – Reynolds number,  $\mu$  - viscosity,  $\rho$  - density,  $v_0$  - initial volume and  $R_0$  undisturbed radius.

The study concerns the analysis of pulsatile flow of blood with rapidly time variant flow. The flow under consideration is associated with the difference between Reynold’s Number at a different radius of the artery leads to the flow stability breaks and forms the vortex at the junction of Bifurcation. This is most possible case when there appears sudden change in the artery diameter. This behavior will be numerically studied using Strauhal’s number which is given by

$$S_t = \frac{f \times R}{\bar{v}}$$

Where  $f$  – frequency,  $R$  - Radius,  $\bar{v}$  - average velocity across the tube (artery)

Here the values of  $\bar{v}$  will be computed using equations of motions at various amplitudes. Further, it is ( $S_t$ ) related to Reynold’s Number which also describes the role in pressure flow determination of the velocity which behaves as oscillatory flow such that Womersly number  $\alpha$  for pulsatile flow is given by

$$\alpha^2 = \frac{R^2 2\pi f}{\nu} = \pi R_e S_t$$

### III. ANALYSIS

Since the arterial wall is elastic, then  $u, v$  and  $p$  are all functions of  $r, z, t$  so that introducing the boundary conditions,

$$u = 0, r = a \text{ (maximum)}, \frac{\partial v}{\partial r} = 0, r = 0 \tag{9}$$

$$u(r, z, t) = v_1(r) e^{\alpha_1 t} \tag{10}$$

$$v(r, z, t) = v_2(r) e^{\beta_1 t} \tag{11}$$

$$p(r, z, t) = p_1(r) e^{\gamma_1 t} \tag{12}$$

Where,  $\alpha_1 = \beta_1 = \gamma_1 = i(n \omega t - y_n z)$

Where  $u$ - velocity,  $v$ - velocity,  $p$  - pressure

Then using equations (9), (10) and (11), the velocity in ICA is obtained as,

$$v_1[ICA] = \begin{cases} (0.875)e^{(0.06061)t} \cos(\omega t - 0.6352) \left(\frac{2.31}{R_1}\right) \left[ R \left(\sqrt{\frac{2\pi f}{\nu}}\right) (0.655 + 0.064R_1^2) + \beta(56.05 + 4444.08R_1^2) \right] \\ (0.875)e^{-(0.0476)t} \cos(\omega t - 0.6352) \left(\frac{2.31}{R_1}\right) \left[ R \left(\sqrt{\frac{2\pi f}{\nu}}\right) (0.655 + 0.064R_1^2) + \beta(56.05 + 4444.08R_1^2) \right] \end{cases} \tag{13}$$

The pressure for ICA is obtained as,

$$p[ICA] = \begin{cases} (2.02125)\omega e^{(0.06061)t} \cos(\omega t - 0.6352) \left(\frac{2.31}{R_1}\right) \left[ R \left(\sqrt{\frac{2\pi f}{\nu}}\right) (0.655 + 0.064R_1^2) \right] \\ (2.02125)\omega e^{-(0.0476)t} \cos(\omega t - 0.6352) \left(\frac{2.31}{R_1}\right) \left[ R \left(\sqrt{\frac{2\pi f}{\nu}}\right) (0.655 + 0.064R_1^2) \right] \end{cases} \tag{14}$$

Solutions of velocity in ICA in response to CCA have been studied for 1/100 sec in 34 time steps. The volumetric flow rate in CCA is given by,

$$Q = \int_0^R 2 \pi r v dr \tag{15}$$

The volumetric flow rate ( $Q$ ) in ICA is given by,

$$Q = \int_0^{R_1} 2 \pi r v_1 dr \tag{16}$$

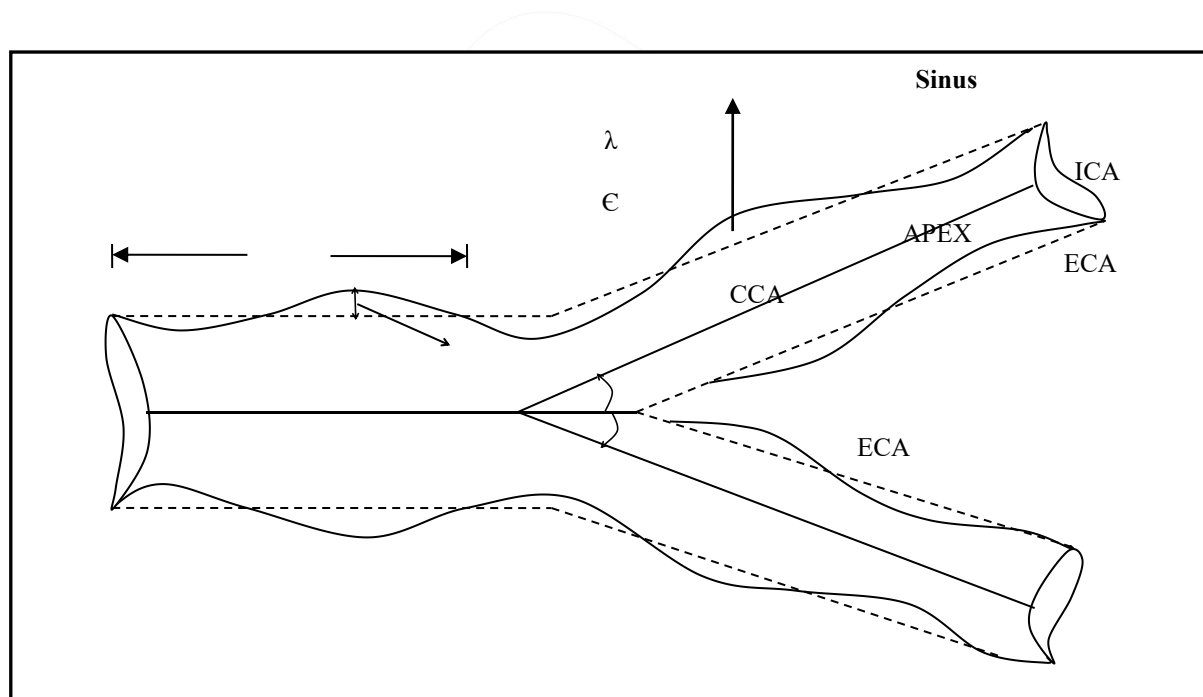
$$Q = \int_0^{R_1} 2\pi r \left\{ \begin{aligned} & (0.875)e^{(0.06061)t} \cos(\omega t - 0.6352) \left(\frac{2.31}{R_1}\right) \left[ R \left(\sqrt{\frac{2\pi f}{\nu}}\right) (0.655 + 0.064R_1^2) + \beta(56.05 + 4444.08R_1^2) \right] \\ & (0.875)e^{-(0.0476)t} \cos(\omega t - 0.6352) \left(\frac{2.31}{R_1}\right) \left[ R \left(\sqrt{\frac{2\pi f}{\nu}}\right) (0.655 + 0.064R_1^2) + \beta(56.05 + 4444.08R_1^2) \right] \end{aligned} \right\} dr \tag{17}$$

Where  $v_1$  value is given by equation (13) for different frequencies. Pressure values are given by equation (14) for different frequencies. The maximum stress occurs at the wall in the disturbed region (sinus or bulb) in ICA. The wave function depicting the distensibility of the wall can be assumed to be a cosine function.

**IV. RESULTS AND DISCUSSION**

Mathematical analysis to compute the velocity, flux and pressure drop at local and global stabilities by introducing Strouhal's number with Newtonian viscosity of blood. A comparison is made with Non Newtonian fluid (blood). Analysis of Velocity and pressure values for CCA and ICA has been done with atherosclerosis plaques in ICA. The velocity profiles in CCA and ICA has been compared. There appears an increase in the peak velocity between the time steps 16 -21 and decrease between the times steps 21- 41.  $\alpha$  and  $\beta$  are taken as constants values for comparing the physiological variations, ranging from the lowest to the highest frequencies (i.e.  $f$  takes from 60 to 130 beats per min) in the downstream of CCA. Radius of the CCA is restricted to 3.3 mm with Reynolds number range 250 - 500. The analysis of the velocity profiles in the carotid sinus both at the apex and in the sinus is made. Prior to the sinus, the flow separation occurs and the region that is affected includes a substantial fraction of the sinus volume

As the fluid passes through the sinus, it again gets accelerated towards the internal carotid artery and thus the wall shear stress increases. Then, at the sinus region the magnitudes of wall shear stress become significantly higher due to the increase of velocity as well as pressure. The flow division ratio is 70%: 30% between the internal and the external carotid artery. The study of velocity and pressure rates has been done for timescales 1, 4, 7 ...100 taken as 1/100 sec. with the varying frequencies.



**Fig. 1: Wall movement of CCA and ICA**

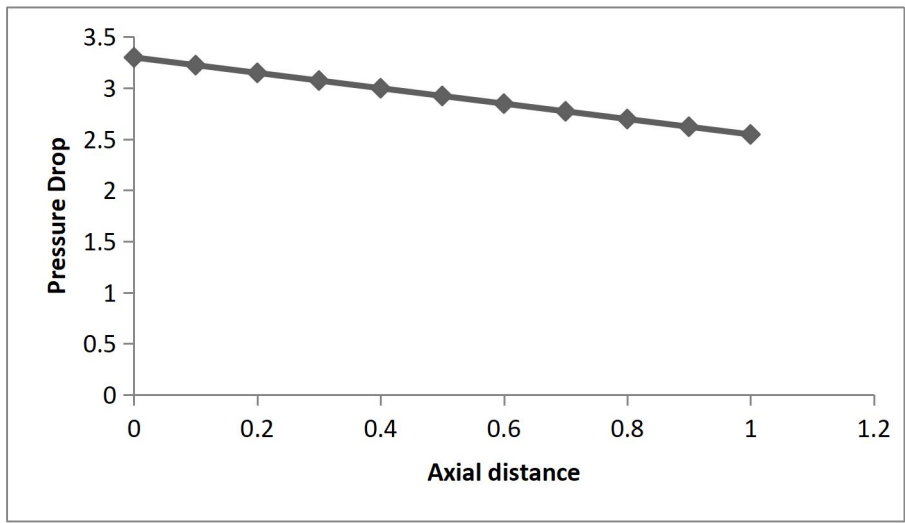


Fig 2: Pressure drop v/s axial distance

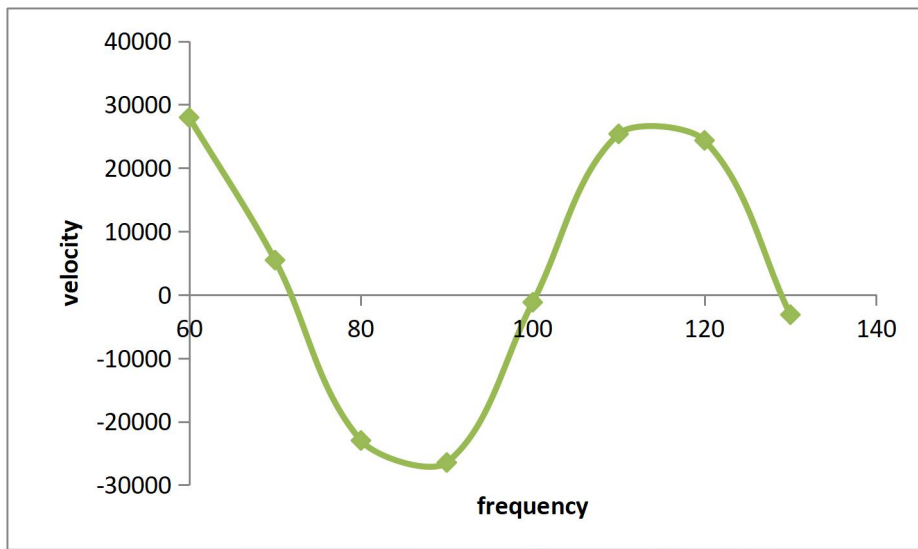


Fig 3: velocity in CCA v/s frequency

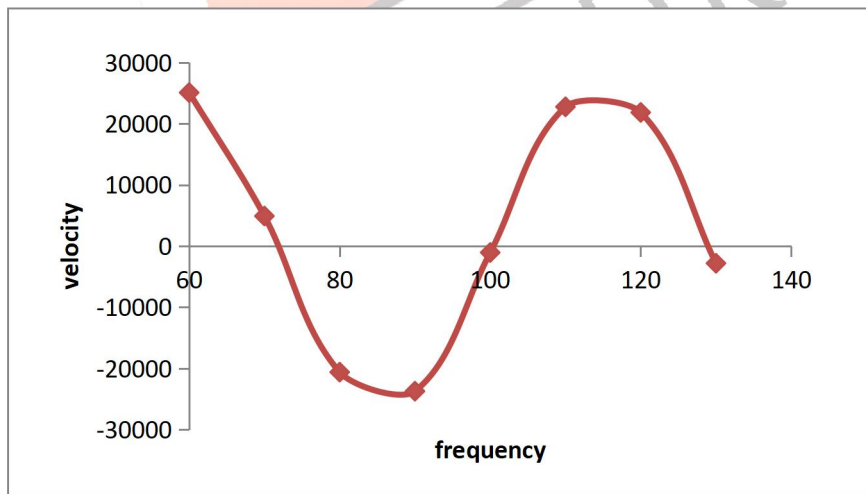


Fig 4: velocity in ICA v/s frequency

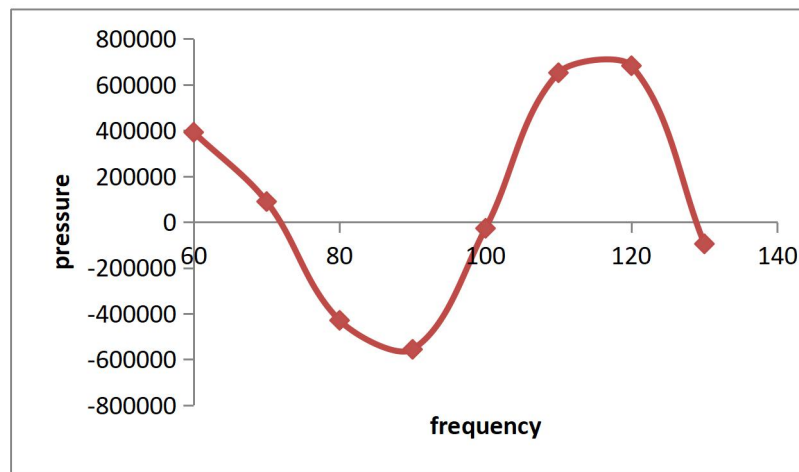


Fig 5: Pressure in CCA v/s frequency

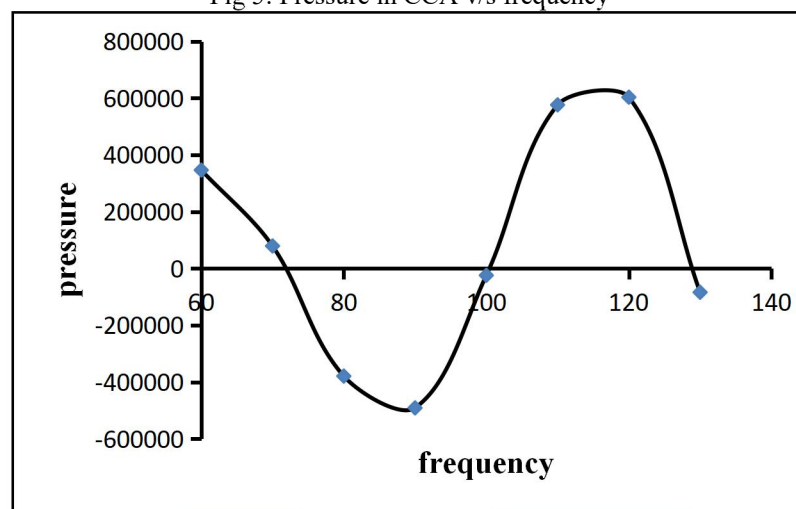


Fig 6: Pressure in ICA v/s frequency

**References**

- [1] Bhardvaj B K, Mabon R F and Giddens [1982], ‘ Steady flow in a model of the human carotid bifurcation part –I – Flow visualization’, J. Biomechanics, Vol. 15, No. 5, 349-362.
- [2] Porenta G, Young D F and Rogge T R [1986], ‘ A finite element model of blood flow in arteries including taper branches and obstructions’, J. Biomechanical Engineering, Vol. 108, 161-167.
- [3] W Kinsner, Y Yan [1990], ‘A model of the carotid vascular system with stenosis at the carotid bifurcation’, Mathematical and computer modeling, Volume 14, PP 582-585.
- [4] Kaluzynski K and Liepsch D [1995], ‘The effect of wall roughness on velocity distribution in a model of the carotid sinus bifurcation- analysis of laser and ultrasound Doppler velocity data’, Technology and health care, Vol. 3, 153-159.
- [5] Botner R, Rappitsch G, Scheidegg M V, Liepsch D, Perktold K and Veisegr P [2000], ‘Hemodynamics in the carotid artery bifurcation a comparison between numerical solution and in-vitro MRI measurments’, J. Biomechanics, Vol. 33, 137-144.
- [6] Basavarajappa [2002], ‘The experimental and numerical analysis of pulsatile flow in carotid artery bifurcations’.
- [7] JaehoonSeong, Baruch B Lieber, Ajay K Wakhloo [2005], ‘Morphological age-dependent development of the human carotid bifurcation’, Journal of Biomechanics, Vol.38, PP 453-465.
- [8] M Mandru, C Ionescu, M Chirita [2009], ‘Modelling Mechanical properties in native and biomimetically formed vascular grafts’, Journal of Bionic Engineering, 6, PP 371-377.
- [9] T Koppl, M Schneider, U Pohl, B Wohlmuth [2014], ‘The influence of an unilateral carotid artery stenosis on brain oxygenation’, Journal of Medical Engineering & Physics, Vol 36, PP 905-914.
- [10] Yakov A Gataulin, Dmitri K Zaitsev, Evgueni M Smirnov, Andrey D Yukhnev [2017], ‘Numerical study of spatial-temporal evolution of the secondary flow in the models of a common carotid artery’, St. Petersburg polytechnical University Journal: Physics and Mathematics 3, PP 1-6.
- [11] S Balamuralitharan and M Radha [2018]. “Bifurcation analysis in SIR epidemic model with treatment”. IOP Conf. Series: Journal of Physics: Conf. Series 1000, 012169.
- [12] Isaac MwangiWangari and Lewi Stone [2018]. “Backward bifurcation and hysteresis in models of recurrent tuberculosis”. PLOS ONE, PP. 1-29.
- [13] Yakin Shu, Jical Huang, Yueping Dong, Yasuhiro Takeuchi [2020], ‘Mathematical modeling and bifurcation analysis of pro – and anti-tumor macrophages’. Applied Mathematical Modeling , Volume 88, pp. 758-773.