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Hemodynamics Effects on Blood Movement through an Artery

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***Abstract*—** Mathematical simulation of blood movement in the arterial system certainly provides the nature and physiology of the creature body. Hemodynamic play the main role to build arterial single or multiple stenoses in the body, which indicate to the abnormalities of the cardio system. Blood functioning, especially in the diseased arteries can be identified by mathematical simulation. Applications of mathematical simulation can be used in surgical planning and configuration of medical devices. The arteries are such organs which can accept the change in varying hemodynamic conditions. In some cases, unusual hemodynamic impacts make an abnormal clinically response. The stenosis becomes the reason for turbulence and flows choking. In the arrival time, the investigation of arterial blood movement will give the prediction of hemodynamic movements in any patient. The advancement of clinical tools to identify the disease and modeling of devices will give better results.

***Keywords*—** Hemodynamic, Stenosis, Artery, Blood Flow.

I. INTRODUCTION

Supply and circulation of essential nutrients also known as a cardio process throughout the body is the most crucial process for all living animals. Recent years have witnessed several deaths due to an abnormality of this process [1]. The fatalities due to cardiac disease may be seen in all the age groups. The reasons may be attributed to changed lifestyle, sedentary or typical office work, lack of proper exercise, adequate sleep as well as eating habits etc. to name a few. Stenosis is one of such complications that deposits of fatty materials, cholesterol, cellular ravage products, calcium as well as other materials build up in the inside part of an artery [2]. This swelling is called plaque. The build-up of heavy tissues as well as calcium in blood vessel walls may become adequate to interfere with blood movement at a fixed point in arteries, like coronary and cerebral arteries. A stenosis is a tapering of the lumen of a blood vessel, and some of the common causes are atherosclerosis, calcification or malformation of valves, and thrombosis caused by tissue trauma. Medically, stenosis refers to the tapering of an artery due to blockage. When either of these arteries is narrowed or blocked, blood and oxygen cannot flow freely to the heart muscle or brain and stenosis develops [3]. When constriction of the vessels occurs, there is a drop in blood pressure across the stenotic lesion. It usually affects large and medium-sized arteries. Some stiffness of arteries generally occurs when a community grew up. Plaques can nurture to significantly lessen the blood's movement through an artery. But most of the clinical damage occurs when they become fragile and rupture. There are many risk factors to generate stenosis such as the use of tobacco, exceed blood pressure and fat etc. Blood movement and its friction with the blood pipes are studies under the hemorheology, an area of science. The creature blood circulatory system is used to bring nutrients and oxygen to the body cells and it leads waste substances away from the same cells. Creature blood is a suspension of cellular components like RBC, WBC, and platelets in electrolyte liquid called plasma [4]. Plasma is having 90 percent of water and only seven percent of major proteins like albumin, lipoprotein, globulin, and fibrinogen. Approximately forty-five percent of volumes are having formed components and rest fifty-five percent of plasma. The ratio between RBC and full volume of blood is called hematocrit. In big and medium tubes, blood is normally considered Newtonian liquid while in smaller tubes; blood is a comprehensive rheological mixture showing Non-Newtonian characteristics like viscous-elasticity or shear thinning. RBC is known as a semisolid particle which augments the viscosity of blood and changes the nature of the fluid. It gives the conclusion that plasma treats like Newtonian fluid while entire blood shows Non- Newtonian characteristic [5].

Healthy blood tubes are very complicated in order. The creature body consists of arteries, veins as well as capillaries tubes for the blood movement. These blood tubes are having different roles so their wall constituents and structures are also different. The wall of big blood tubes has circumferentially layered structure like intima, adventitia, and media [6].

II. PRESSURE WAVE PROPAGATION

It is a regular process to generate pressure by the flowing blood on the walls of tubes. Several departments of creature body are helping to the regulation of blood pressure. Arteries walls are having more blood pressure compared to veins. In the time of abnormalities, the blood pressure in the creature arteries may arise. Pumping of the heart is forming pressure wave in creature body. The pressure wave may very useful to identify a diseased area in creature body. To understand blood movement in arteries, we have to know that pressure wave propagation carries viscous and incompressible fluid through a stressed tube. In this process, fluid is assumed Newtonian and tube is considered isotropic and elastic. Indeed, the thick arterial wall gives more realistic results compared to a thin wall. In this study, the Non-linear terms are not considered for the blood movement. It is also investigated that the longitudinal displacement is very large compared to the longitudinal oscillation of the tube wall [7]. In the normal condition, PWV is calculated by

$$PWV = \frac{L}{\Delta t} \tag{1}$$

Here L as well as Δt is the distance between two points and time delay of the waveform respectively.
PWV is mostly applied to find a vascular disease like atherosclerosis. For the big and straight flexible pipe [according to the Moens-Korteweg equation]

$$PWV = \sqrt{\frac{Eh}{2\rho r_i}} \tag{2}$$

Where E has assumed Young's modulus of an arterial wall, the wall thickness is taken h, ρ is blood density, an internal radius of an artery is r_i. For thick- walled pipes, Moens-Korteweg equation is modified by calculating the strain on the middle wall of the pipe.

$$PWV' = \sqrt{\frac{Eh}{2\rho\left(r_i + \frac{h}{2}\right)}} \tag{3}$$

Modified Moens-Korteweg solution is applied once again in the occurrence of movement

$$PWV'' = \sqrt{\frac{Eh}{2\rho\left(r_i + \frac{h}{2}\right)}} + U \tag{4}$$

Here U is said to be the cross-sectional mean velocity of the liquid. It is identified that Young modulus (E) is directly proportional to PWV. This relation is applicable for long, straight and flexible homogeneous pipe only. The wall of pipe must be very thin with non-viscous liquid [8]. The anatomy and constitution of the artery differ from one place to another.

TABLE 1

Young's modulus and the wall thickness of the artery model

Young's Modulus E (MPa)	0.5	1.0	2.0
Wall Thickness h (m.m.)	1.5	2.0	4.0

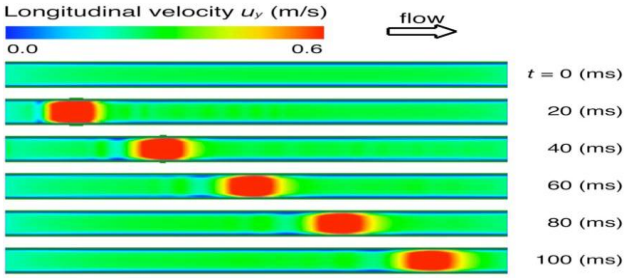

Figure1. Longitudinal velocity u_y in the uniform pipe with Young's modulus of 1.0 MPa along with the thickness of 2.0 mm at the plane $x = 0$.

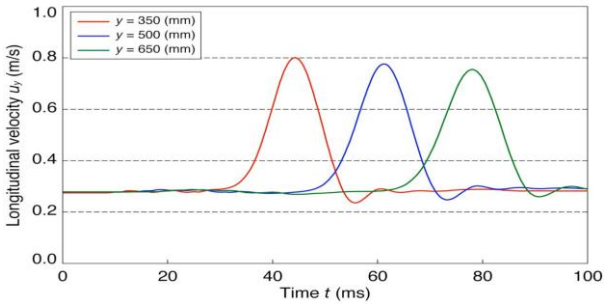

Figure2. Middle-line speed waveforms at distinct locations with Young's modulus of 1.0 MPa along with wall thickness of 2.0 mm.

III. BLOOD FLOW MODELS

There are five Non-Newtonian and one Newtonian model for the study of shear stress distribution through an artery. In Power law as well as Walburn-Schneck model, viscosity reduces due to raising in strain up to 226.5 per second while viscosity goes up to a maximum shear rate in Carreau and Casson model. In some cases, Generalized Power law model behaves differently to the power law model. In these models, blood is considered incompressible. Density is taken approximately 1051 kg per m^3 and these models govern by continuity and Navier-Stoke equation. Artery must be rigid and equations are resolve by finite volume solution. To solve flow equations, some boundary conditions are taken. There is no slip condition works at the boundary wall but it is totally different for the outlet. The velocity data is supposed to be the paraboloidal shape. The nature of wall shear stress in a specific artery for specific inlet velocity is the same for Newtonian as well as Non-Newtonian models. The different inlet velocities affect the wall shear stress. The magnitude

differences of wall shear stress are important at low inlet velocity. Power-law models are used to make wall shear stress at little inlet velocity and underestimate at high inlet velocity. The Walburn-Schneck system ignores wall shear stress at up inlet velocity and Newtonian model ignores to wall shear stress at little inlet velocity. Due to these facts, the Power law, as well as Walburn Schneck, is not perfect for blood viscosity modeling compare to others. Newtonian model is good in average range to high shear rate. The Generalized model gives a better outcome in wall shear stress compared to the Newtonian model and majorly plays a very big role in low inlet velocities.

Pressure and flow have pulsatile flow which is varying in various section of the arterial system. The nature of fluid mechanics describes the connection between pressure wave and blood movement. The functioning of the vascular structure may understand by one dimension of modeling of flow. Windkessel model is the example of one dimension which is used to find increasing and decreasing of the movement and pressure waveforms. In this model, the compliant aorta works like a capacitor during systole to collect blood. Thereafter flexible aorta discharges the collected blood to smaller arteries and organs during diastole. The waveform and pressure measured by this model are very close. Thereafter a lot of investigators have given more advanced models. Lumped parameter method is useful to find waveforms and work by heart on the time of ejection. This approach is also useful to determine variations of blood movement distribution at branch points as well as tumor cases. It is also useful to find pulse wave propagation as well as wave speed. To know the outlet boundary conditions, these models may be worth full [9].

IV. FLUID-STRUCTURE INTERACTION

It is a pairing between the principles of fluid dynamics as well as structural mechanics. When a fluid movement encounters then strain and stress are applied on the solid object then fluid structure goes to deformation. Sometimes deformation may be very small or sometimes very large. It relies on the velocity and pressure as well as sometimes on the characteristics of the genuine structure. If deformations are very small along with the slow variation of time then the fluid movement will not be affected by deformation. If a variation is fast then small deformations lead to pressure waves in blood.

In mathematical design, we need either exploit or avoid the main effects of fluid-structure interactions. The peristaltic pump is the example of it. A peristaltic pump has to exploit significant structure deformation to tenderly blood pump without harming living cells.

The finite element simulation is useful to find fluid interaction in the aortic valve. For this process, blood must be Newtonian. Replica of the left ventricle, as well as aortic valve, is useful for this study. Firstly simulation of the left ventricle is obtained to get fluid-structure interaction. With the assistance of these statistics, the aortic valve is designed. In the result, the pressure difference is uniform in the valve leaflet. FSI is also useful to find modeling of one and two ruptured aneurysms and sometimes modeling of an aneurysm is possible in middle cerebral artery. Blood flow is taken laminar and incompressible. This flow relies on the Navier-Stoke equation. The mechanism of the artery wall is obtained by force equilibrium equations. The modeling of the artery wall is possible with linearly flexible material and finite strain. The shape of one and two an aneurysm plays a very big role in fluid-structure interaction. The periodic and incompressible movement through a narrowed vessel is also modeled by time-periodic function in fluid interaction model. This time a periodic method is obtained for curved, straight and bifurcating arteries. This time coupling method is compared with weakly coupled arteries. It shows that periodic coupled model gives better result compared to weakly coupled approach [10].

V. ENTRANCE REGIONS

A pressure reservoir brings flow from the heart to small tubes. At the starting point of some arteries, the movement is not completely developed. It is equivalent to the potential core as well as developed boundary layer at the wall. The velocity profile is maximum in the centre and centreline speed of blood accelerates the border layer retards speed close the wall. Unsteady movement concerned to entrance region relies on Womersley parameter and Reynolds number. For less Womersley parameter, the highest entrance length is equivalent to peak movement and it relies on the Reynolds number. For maximum Womersley parameter, border layer development is faster and entrance length is continuous during the cycle [11].

The thickness of boundary-layer develops from the entrance (δ_1) if u is the concerned instantaneous main velocity. The oscillatory border-layer thickness (δ_2) is equal to 6.5 $(n/w)^{1/2}$ for oscillation of angular frequency. Then unsteady entrance length l_1:

$$I_1 = 3.4u/w$$

VI. CURVED TUBES

Movement in a curved blood vessel was observed by Dean (1928). He has taken a parameter concerning the centrifugal forces to viscous forces. It is a connection between Reynolds number and a nondimensional ratio of pipe radius and curvature.

$$Dean\,number = (2\delta)^{1/2}.4\,\mathrm{Re}$$

$$\delta = \frac{Radius\ of\ pipe\ cross\ section}{Radius\ of\ curvature\ at\ centerline}$$

The aortic arch is having two fundamental flow circumstances. If movement in the entrance of pipe is not full then the core of fluid in curve behaves like potential vortex along velocity towards to inner wall [12].

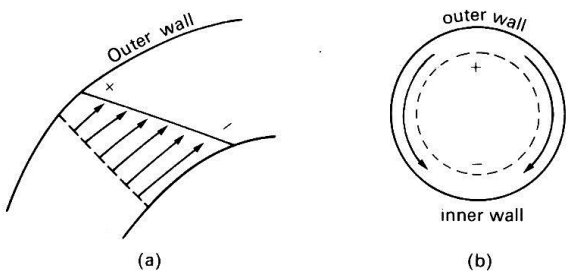

Figure3. (a) Velocity profile in a curved blood vessel near the entrance (ignoring the boundary layer). + means high pressure, - means low pressure. (b) The pressure difference from + to - drives secondary motions in the boundary layers, as indicated by the arrows.

VII. SEPARATED FLOWS

Some places in the arteries system are having expanded regions. Sometimes cross sections area and the axes may enlarge at aneurysms and sinuses. The change in cross-sectional area may get by continuity equation as

$$\bar{u}\,A = Q(t) \tag{5}$$

Then the unsteady Euler equation is used to find local pressure changes for area enlargement

$$\frac{\partial \bar{u}}{\partial t} + u\frac{\partial \bar{u}}{\partial x} = -\frac{1}{\rho}\frac{\partial p}{\partial x} \tag{6}$$

After rearrangement equation becomes

$$\frac{\partial p}{\partial x} = -\frac{\rho}{A}\frac{dQ}{dt} + \frac{\rho Q^2}{A^3}\frac{dA}{dx} \tag{7}$$

From the equation (7), we can see that the required gradient exists during the acceleration of flow and converging of the area. If the area is diverging when movement is decelerating then advanced gradient may exist [13].

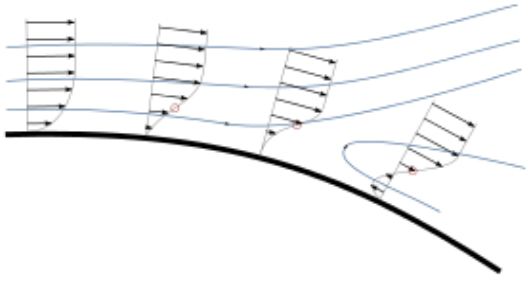

Figure4. Separated flow

VIII. SHEAR STRESS

During the blood movement through endothelium, shear stress makes the retarded flow

$$\tau = \mu\frac{du}{dr} \tag{8}$$

Shear stress in a straight pipe is obtained as

$$\tau_{wall} = \frac{32\mu Q}{\pi D^3} \tag{9}$$

In some circumstances when the lumen isn't circular or blood movement is skewed at branch points, shear stress varies widely. It doesn't easily work for pulsatile flow. The velocity and gradient must be calculated near the wall, which seems technically difficult. Gradient depends on the velocity profile and distance from the wall. An alternative way is used with shear stress sensor which evaluates the heat transfer between closed points on the wall [14].

IX. HOOP STRESS

Arteries as well as veins are having mean pressure approximately $100\,mmHg$ and $10\,mmHg$ respectively. It distends the pipe like a big balloon, so the pipe must resist this force with hoop stress. The hoop stress can be computed by Laplace rule as [15]

Resisting force = bursting force.

Hoops stress× resisting area = P ×d×l

Assuming, F (h/c) = hoops stress

F(h/c)×2×t×l = P×d×l

F(h/c)= P d/2t

$$\sigma = \frac{PR}{t} \tag{10}$$

Here R and t are radius and wall thickness respectively.

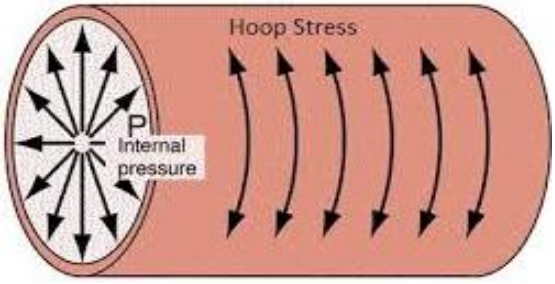

Figure5. Hoop Stress

X. BIOLOGICAL RESPONSES TO HEMODYNAMICS

Artery reacts during the blood movement in mechanical stress. Many parameters are required to sustain the regular performance of the circulatory system. The reaction of arteries to hemodynamic nature may generate some pathological ailment.

A. HEMOSTASIS

Hemostasis is concerned with bleeding. Trauma is normal and the creature body must deal with it. It occurs in a very short duration of a millisecond to one minute. When a blood pipe is damaged through trauma, blood comes out through the hole. In the hemodynamic process, hemostasis becomes through platelet and adherence. Some studies indicate that platelet to platelet collection depends on the shear stress. To understand hemostasis, we have to know very well the effect of hemodynamics.

B. DILATION AND CONTRACTION

An artery is used to give the response in hemodynamic minute to minute. The blood pipes must be able to adapt the demands and conditions of blood pressure as well as movement change. Systemic movement adaptation happens due to many mechanisms like controlling the heartbeat, flow channelize by autoregulation and blood quantity control y kidneys. Arteries are used to dilate when augmenting of movement through endothelial releases nitrous oxide. The hemodynamic parameter plays a very big role to release this nitrous oxide. Blood pipes contract

in short duration for hormonal stimulation. Contraction is normally happened to maintain systemic vascular resistance, intravascular blood quantity, and venous pooling.

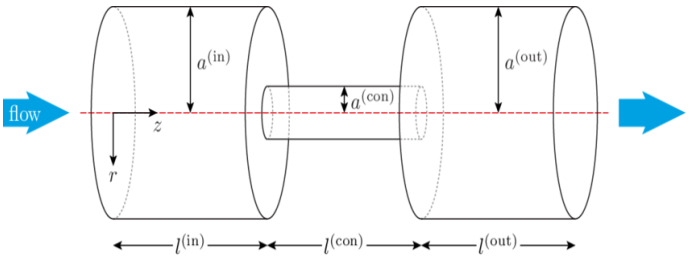

Figure6. Schematic drawing of contraction-expansion movement geometry

C. ADAPTION AND REMODELING

Arteries accept adaption for long-term augments or decreases in wall stress. The consequence to augmented wall stress is to vasodilate as well as then remodel to a big diameter along with the similar arteries structure. Normal wall stress makes remodel to normal blood pipe of smaller diameter. On a long duration of months to years, arteries make remodel their concern intima as well as media layers. The medial thickness depends on hoop stress as well as nutrition.

D. PATHOLOGY

Long-term hypertension generates a medial thickening of blood pipes. Hypertension becomes a reason for stiff and thick arteries that may change the blood flow behavior. Normal fluctuations in blood movement are not possible. Vascular grafts make a thickness that becomes the reason for stenosis. Vascular grafts are having a large diameter which stimulates an, unlike response. Atherosclerosis disease takes decades. This disease develops in systemic vasculature, carotid and coronary arteries, superficial femoral artery, and abdominal aorta. The wall stress is minimum and fluctuates between both directions during the cardiac cycle in these arteries. Mostly intimal thickness occurs where the normal wall stress is less than 10 dynes per centimeter square. Here patterns are having an inverse relationship between intimal hyperplasia and arterial adaptation. The consequences of hemodynamic on mass transfer must not be neglected. Mostly active molecules are used to convict from one place to another. These molecules are nutrients, growth factor or wastes. Systemic hormones go to arteries through convection and may diffuse by the wall. In this condition, intima is a big obstacle [16].

XI. HEMODYNAMICS OF STENOSIS

Due to some conditions, arteries become acutely diseased. Arterial lumen has a restricted layer over a one-centimeter distance. Medically this constriction is known as stenosis. Hydrodynamic investigations of these movement constrictors may provide some data of pressure drop as well as a nonrecoverable head loss by separation and turbulence. Stenosis is defined in percent by

$$Percent\ stenosis = \frac{D_1 - D_2}{D_1} \times 100\,\%$$

Stenosis movements have been well defined in various calculations. Movement separation happens in expansion area at Reynolds digits for a 70 percent stenosis. In this case, a hard shear layer becomes between the central jet as well as a recirculation area. If the stenosis is greater than 75 percent than mostly arterial mechanism makes a slow movement. The turbulence of stenosis is very big for 75 percent and makes a proper resistance. At the maximum level of stenosis, turbulence is acute and responsible for 80 % pressure loss [17].

XII. CONCLUSION

Non-Newtonian nature of blood in small arteries will give more accurate results. Prediction, as well as the impact of the pressure wave, play a very big to identify clinically diagnosis area. Arterial hemodynamics may be resolved to take an unsteady movement. Infected arteries may generate exceed the level of turbulence and choked- movement circumstances

in which pipes can collapse. Fluid mechanics plays a very big role in detection and remedies of arterial disease. The quantity of stress, as well as mass change at the liquid –wall interface, are major hemodynamic factors affecting the medical responses. The development of stenosis is the main biological response. A combination of movement pressure and cardiovascular stenosis makes a serious problem. So some medical devices are being developed to maintain and repair stenosis. Hence, we can say that fluid mechanics will play a big role to identify future cardiovascular diseases.

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