A MATHEMATICAL THEORY FOR BLOOD FLOW DYNAMICS IN THE ARTERIAL SYSTEM an induction of blood flow velocity.

A MATHEMATICAL THEORY FOR BLOOD FLOW DYNAMICS IN THE ARTERIAL SYSTEM.

an induction of blood flow velocity.

Hirohumi HIRAYAMA, Kohichi ONO, Hisakazu, YASUDA

Summary

A theoretical expansion of mathematical models of the cardiovascular system are developed. We established a distributed parameter model of the arterial system. In this paper we have deduced the blood flow velocities in the longitudinal and radical direction based mainly on the Womersley theory. Neglecting the non-linear terms (the convective acceleration terms) in the Navier-Stokes equation and setting linear cyclic solutions, the N-S equations were reduced to the Bessel type ordinary differential equations. By utilyzing the Stokes stream function, the equation which input pressure satisfy was proved to be a Bessel type differrential equation. Applying the Bessel type pressure function to the linearlyzed N-S equation, a strict form of the solution of the blood flow velocities were obtained. these solutions were confirmed to satisfy the conservative law of mass.

To ensure whether these solution satisfy the Stokes stream function another process was used to obtain the blood flow velocities. Turning to the stream function and differentiating directly of these functions also induced a series of solutions which are identical with the solution that were obtained by solving the Bessel type N-S equation. By these strict mathematical process, linear solutions of the blood flow velocities were obtained. To simplyfy the system and problems we made some assumptions and we have discussed the validity of these assumptions within the range we concern.

Introduction

It is important to correlate the biological phenomenone and their interactions quantitatively. In such a stand point, the cardiovascular system is one of the most suitable subject for such analysis. Especially to represent the pulse wave transmission phenomenone in human arterial system gives much advantages for understanding the control mechanisms of the circulatory system. Furthermore in the pathophysiological state especially for the congestive heart failure or the transplantation of the heart, it should be analyzed that the interaction and feed back control mechanisms of pulse wave conduction which should appear dynamically between the heart and the peripheral circulation.

To satisfy such requirement, mathematical or physical models of the circulatory system have

Muroran Institute of Technology. Deaprtmant of Caridovascular Medicine.Hokkaido univ.

been presented. For the arterial system, beginning with Witzig (1914), the essential and pioneering work of the elastic tube theory have been expanded by Womersley (1958) [1]. On the other hand the physical electrical model also have been constructed elaborously at the same time and the basic model was completed by the group of Westerhoff and Nordergraaf (1967) [2].

The mathematical model can be classified into 3 categories on the basic of common feautuers of assumptions. The first group is the thin walled model which is based on the membrane theory. Morgan (1954), Womersley (1954) [3], Klip (1962), Atabeck (1968), and Chow (1967) participated in this type of model.

The second group is the thick walled model. Klipp (1967), Mirsky (1967), Cox (1969), Jager (1966) [4], Whirlow (1965), concerned this field of model. The last group is the longitudinal tethering model and is consequently indentical with the rigid tube model. Witizg (1914), Womersley (1958), Taylor (1959), Jones (1969) dealed with this model.

Althought precise and complicated models have been presented by these researchers, they concerned only with the pulse wave velocity or the transmission efficiency. What we should make clear is how transmission phenomenone can be represented or revealed realistically. Yet these problems are solved.

About for the physical models, kind of electrical or hydrodynamical analog have been constructed. By connecting many condencers or registances, the blood flow waves can be simulated in the arbitrary precision. To increase the approximation, one can reinforce the elements of the circuit and further complex circuit can be easily constructed. However the biophysical significances of each elements embeded in the circuit would become obscure [5].

Because of the histhero mentioned grounds, there exists a reasonable necessity to establish a comprehensive and easier recongnizible mathematical model. It is a vital necessity for the purpose of analyzing the effects of changes of the arterial wall and blood properties on the arterial blood flow.

In the series of these papers, we have constructed 3 basic models about the cardiovascular system, the distributed parameter model of the arterial system, the exponential paramerter model of the aortic arch, the lumped circuit model of the total systemic circulation.

In these three models we firstly show the theoretical expansion about the distributed parameter model of the peripheral arterial system which is based mainly on the transmission line theory in 4 steps.

Then we reveal how it does express the pulse wave transmission phenomenone in time and space domain realistically and the effects of changes in the biophysical parameters of the arterial

wall and blood on the blood flow parameters. In this paper as the first step for the mathematical expansion, we have deduced the strikt form of the blood flow velocities from the linearlyzed Navier-Stokes equations by two different solution process.

MATHEMATICAL EXPANSION-1

To represent the pulsatile flow through a distensible tube mathematically,it is necessary to obtain sets of equations which include not only the blood properties itsself but also the mechanical properties of arterial wall

For the purpose of such requirements. the equation about the blood flow dynamics and wall motion should be given independently. Then those equations must be associated by setting the adequate boundary conditions. In this chapter we reduce the blood flo v velocities from the Navier-Stokes fluid dynamic equations.

Before solving the equations, we have made following assumptions about the blood properties and the geometric characters of the vessels.

1. The blood is Newtonian and incompressive.
2. The blood viscosity is independent of blood shear rate. haematcrit, body temperature, blood flow velocity nor internal radius of the artery.
3. The blood flow contains only laminar flow. The tangential blood flow velocity is very small and the secondary flow. nor turbulence exists.
4. The abnormal viscosity does not exists.
5. The effects of the entry zone are negligible.
6. The sllipage between the blood and the vessel wall at the innner surface of the wall does not exists.
7. The vessel is straight, cylindrical, and axisymmetric.
8. The biophysical properties of the vessel wall are constant and independent of the distance from the entry zone.
9. There exists no tapering of the vessel and no leakage flow.
10. The effect of the gravity is negligible.

l] The fluid dynamic equations of the blood flow.

The movements of the blood in the closed space especially in the cylindrical tube as a vessel are expressed in the Navier Stokes equations. In the cylindrical coordinates, the blood flow velocities satisfy following equations.

For the longitudinal direction

$$\rho\left(\frac{\alpha Vz}{\alpha t}+Vr\frac{\alpha Vz}{\alpha r}+Vz\frac{\alpha Vz}{\alpha z}\right)=\frac{-\alpha P}{\alpha z}+\mu\left(\frac{\alpha^2 Vz}{\alpha r^2}+\frac{1}{r}\frac{\alpha Vz}{\alpha r}+\frac{\alpha^2 Vz}{\alpha z^2}\right) \quad (1)$$

For the radial direction

$$\rho\left(\frac{\alpha Vr}{\alpha t}+Vr\frac{\alpha Vr}{\alpha r}+Vz\frac{\alpha Vr}{\alpha z}\right)=-\frac{\alpha P}{\alpha r}+\mu\left(\frac{\alpha^2 Vr}{\alpha r^2}+\frac{1}{r}\frac{\alpha Vr}{\alpha r}+\frac{\alpha^2 Vr}{\alpha z^2}-\frac{Vr}{r^2}\right) \quad (2)$$

The variables and parameters are defind as followings

Vz : the instantaneous blood flow velocity parallel to the vessel axis. (the longitudinal blood flow velocity.)

Vr : the instantaneous blood flow velocity along the radial coordinates. (the radial blood flow velocity.)

Z : the longitudinal space coordinates.

r : the radial space coordinates.

p : the internal pressure.

ρ : the blood density.

μ : the blood viscosity.

The left sides of the equation 1,2 are in the form of unit mass(ρ) multiplied by the acceleration DV/Dt which mean the internal force in the longitudinal direction and the radial direction respectively. To balance such forces, two forces are given in the right hand of these equations. these are the pressure gradient along the axis $\alpha p/\alpha z$ and along the radius $\alpha p/\alpha r$.

Furthermore the viscous retardation force (the frictional force) contributes. In general, for the case of dynamically moving fluid, the stress changes parallely with the velocity of the deformation of the fluid, that is the shear rate of the blood flow.

Assuming there exists no leakage flow, the conservative law stands.

Then continuity equation is given as following.

$$\frac{\alpha\rho}{\alpha t}+\frac{1}{r}\frac{\alpha(\rho Vr*r)}{\alpha r}+\frac{1}{r}\frac{\alpha(\rho V\theta)}{\alpha\theta}+\frac{\alpha(\rho Vz)}{\alpha z}=0 \quad (3)$$

We also assume the incompressibillty of the blood, the blood density does not change with time. Then the eq 3 is reduced as following.

$$\frac{\alpha Vr}{\alpha r}+\frac{Vr}{r}+\frac{\alpha Vz}{\alpha z}=0 \quad (4)$$

Since we concern only in the linear system, we seek the linear solutions for this system. Therefore these above mentioned non linear partial differential equations should be linealized. Assuming that the effects of the convective acceleration terms such as

$$Vr\frac{\alpha Vz}{\alpha r}, Vz\frac{\alpha Vz}{\alpha z}, Vr\frac{\alpha Vr}{\alpha r}, Vz\frac{\alpha Vr}{\alpha z}$$

on the flow velocity are negligible, we linealized the equ 1 2 to following form.

$$\frac{\alpha Vz}{\alpha t} = -\frac{1}{\rho}\frac{\alpha P}{\alpha z} + \frac{\mu}{\rho}\left(\frac{\alpha^2 Vz}{\alpha z^2} + \frac{1}{r}\frac{\alpha Vz}{\alpha r} + \frac{\alpha^2 Vz}{\alpha r^2}\right) \tag{5}$$

$$\frac{\alpha Vr}{\alpha t} = -\frac{1}{\rho}\frac{\alpha P}{\alpha r} + \frac{\mu}{\rho}\left(\frac{\alpha^2 Vr}{\alpha z^2} + \frac{1}{r}\frac{\alpha Vr}{\alpha r} + \frac{\alpha^2 Vr}{\alpha r^2} - \frac{Vr}{r^2}\right) \tag{6}$$

A] The induction of the equation which satisfy the input pressure P.

Before solving the linealized N-S equations, we should obtain the functional form of the input pressure P.

Define the Stokess stream function ψ which satisfy the continuity equation 3 as following. [6]

$$Vz = -\frac{1}{r}\frac{\alpha\psi}{\alpha r} \tag{7}$$

$$Vr = \frac{1}{r}\frac{\alpha\psi}{\alpha z} \tag{8}$$

For the sake of obtaining the relation between and P, we input equ 7 into equ 5, then

$$\frac{\alpha}{\alpha t}\left(\frac{-1}{r}\frac{\alpha\psi}{\alpha r}\right) = \frac{\mu}{\rho}\left(\frac{\alpha^2}{\alpha z^2}\left(\frac{-1}{r}\frac{\alpha\psi}{\alpha r}\right) + \frac{1}{r}\frac{\alpha}{\alpha r}\left(\frac{-1}{r}\frac{\alpha\psi}{\alpha r}\right) + \frac{\alpha^2}{\alpha r^2}\left(\frac{-1}{r}\frac{\alpha\psi}{\alpha r}\right)\right)$$

$$-\frac{1}{\rho}\frac{\alpha P}{\alpha z}$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha}{\alpha r}\left(-\frac{\alpha^2\psi}{\alpha z^2}\right) + \frac{1}{r}\left(\frac{1}{r^2}\frac{\alpha\psi}{\alpha r} - \frac{1}{r}\frac{\alpha^2\psi}{\alpha r^2}\right) + \frac{\alpha}{\alpha r}\left(\frac{1}{r^2}\frac{\alpha\psi}{\alpha r} - \frac{1}{r}\frac{\alpha^2\psi}{\alpha r^2}\right)\right)$$

$$-\frac{1}{\rho}\frac{\alpha P}{\alpha z}$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha}{\alpha r}\left(-\frac{\alpha^2\psi}{\alpha z^2}\right)+\frac{1}{r^3}\frac{\alpha\psi}{\alpha r}-\frac{1}{r^2}\frac{\alpha^2\psi}{\alpha r^2}-\frac{2}{r^3}\frac{\alpha\psi}{\alpha r}+\frac{2}{r^2}\frac{\alpha^2\psi}{\alpha r^2}-\frac{1}{r}\frac{\alpha^3\psi}{\alpha r^3}\right)$$

$$-\frac{1}{\rho}\frac{\alpha P}{\alpha z}$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha}{\alpha r}\left(-\frac{\alpha^2\psi}{\alpha z^2}\right)-\frac{1}{r^3}\frac{\alpha\psi}{\alpha r}+\frac{1}{r^2}\frac{\alpha^2\psi}{\alpha r^2}-\frac{1}{r}\frac{\alpha^3\psi}{\alpha r^3}\right)-\frac{1}{\rho}\frac{\alpha P}{\alpha z}$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha}{\alpha r}\left(-\frac{\alpha^2\psi}{\alpha z^2}\right)+\frac{1}{r}\frac{\alpha}{\alpha r}\left(\frac{1}{r}\frac{\alpha\psi}{\alpha r}\right)-\frac{1}{r}\frac{\alpha}{\alpha r}\left(\frac{\alpha^2\psi}{\alpha r^2}\right)\right)-\frac{1}{\rho}\frac{\alpha P}{\alpha z}$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha}{\alpha r}\left(-\frac{\alpha^2\psi}{\alpha z^2}+\frac{1}{r}\frac{\alpha\psi}{\alpha r}-\frac{\alpha^2\psi}{\alpha r^2}\right)\right)-\frac{1}{\rho}\frac{\alpha P}{\alpha z}$$

We also input equ 8 into equ 6, then,

$$\frac{\alpha}{\alpha t}\left(\frac{1}{r}\frac{\alpha\psi}{\alpha z}\right)=\frac{\mu}{\rho}\left(\frac{\alpha^2}{\alpha z^2}\left(\frac{1}{r}\frac{\alpha\psi}{\alpha z}\right)+\frac{1}{r}\frac{\alpha}{\alpha r}\left(\frac{1}{r}\frac{\alpha\psi}{\alpha z}\right)+\frac{\alpha^2}{\alpha r^2}\left(\frac{1}{r}\frac{\alpha\psi}{\alpha z}\right)-\frac{1}{r^3}\frac{\alpha\psi}{\alpha z}\right)$$

$$-\frac{1}{\rho}\frac{\alpha P}{\alpha r}$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha^3\psi}{\alpha z^3}+\frac{1}{r}\left(-\frac{1}{r^2}\frac{\alpha\psi}{\alpha z}+\frac{1}{r}\frac{\alpha^2\psi}{\alpha r\alpha z}\right)+\frac{\alpha}{\alpha r}\left(-\frac{1}{r^2}\frac{\alpha\psi}{\alpha z}+\frac{1}{r}\frac{\alpha^2\psi}{\alpha r\alpha z}\right)\right.$$

$$\left.-\frac{1}{r^3}\frac{\alpha\psi}{\alpha z}\right)-\frac{1}{\rho}\frac{\alpha P}{\alpha r}$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha^3\psi}{\alpha z^3}-\frac{1}{r^3}\frac{\alpha\psi}{\alpha z}+\frac{1}{r^2}\frac{\alpha^2\psi}{\alpha r\alpha z}+\frac{2}{r^3}\frac{\alpha\psi}{\alpha z}-\frac{1}{r^2}\frac{\alpha^2\psi}{\alpha r\alpha z}-\frac{1}{r^2}\frac{\alpha^2\psi}{\alpha r\alpha z}\right.$$

$$\left.+\frac{1}{r}\frac{\alpha^3\psi}{\alpha z\alpha r^2}-\frac{1}{r^3}\frac{\alpha\phi}{\alpha z}\right)$$

$$= \frac{\mu}{\rho}\left(\frac{1}{r}\frac{\alpha^3\psi}{\alpha z^3}-\frac{1}{r^2}\frac{\alpha^2\psi}{\alpha z\alpha r}+\frac{1}{r}\frac{\alpha^3\psi}{\alpha z\alpha r^2}\right)-\frac{1}{\rho}\frac{\alpha P}{\alpha r}$$

$$= \frac{\mu}{\rho}\left(\frac{\alpha}{\alpha z}\left(\frac{\alpha^2\psi}{\alpha z^2} - \frac{1}{r}\frac{\alpha\psi}{\alpha r} + \frac{\alpha^2\psi}{\alpha r^2}\right)\right) - \frac{1}{\rho}\frac{\alpha P}{\alpha r}$$

then we get equ 9, equ 10,

$$-\frac{1}{r}\frac{\alpha^2\psi}{\alpha r \alpha t} = \frac{\mu}{\rho}\frac{1}{r}\frac{\alpha}{\alpha r}\left(-\frac{\alpha^2\psi}{\alpha z^2} + \frac{1}{r}\frac{\alpha\psi}{\alpha r} + \frac{\alpha^2\psi}{\alpha r^2}\right) - \frac{1}{\rho}\frac{\alpha P}{\alpha z} \tag{9}$$

$$\frac{1}{r}\frac{\alpha^2\psi}{\alpha z \alpha t} = \frac{\mu}{\rho}\frac{1}{r}\frac{\alpha}{\alpha z}\left(\frac{\alpha^2\psi}{\alpha z^2} - \frac{1}{r}\frac{\alpha\psi}{\alpha r} + \frac{\alpha^2\psi}{\alpha r^2}\right) - \frac{1}{\rho}\frac{\alpha P}{\alpha r} \tag{10}$$

Next step we eliminate P in equ 5, equ 6 by differentiating equ 5 with respect to r and diffrentiating equ 6 with respect to z. firstly we differentiate equ 5 from left side.

$$\frac{\alpha}{\alpha r}\left(\frac{\alpha Vz}{\alpha t}\right) = -\frac{1}{\rho}\frac{\alpha}{\alpha r}\left(\frac{\alpha P}{\alpha z}\right) + \frac{\mu}{\rho}\left(\frac{\alpha^3 Vz}{\alpha z^2 \alpha r} + \frac{\alpha^3 Vz}{\alpha r^3} - \frac{1}{r^2}\frac{\alpha Vz}{\alpha r} + \frac{1}{r}\frac{\alpha^2 Vz}{\alpha r^2}\right)$$

$$= \frac{-1}{\rho}\frac{\alpha}{\alpha r}\left(\frac{\alpha P}{\alpha z}\right) + \frac{\mu}{\rho}\left(\frac{\alpha^2}{\alpha z^2} + \frac{1}{r}\frac{\alpha}{\alpha r} + \frac{\alpha^2}{\alpha r^2} - \frac{1}{r^2}\right)\cdot\frac{\alpha Vz}{\alpha r} \tag{11}$$

Differentiate equ 6 from lift side.

$$\frac{\alpha}{\alpha z}\left(\frac{\alpha Vr}{\alpha t}\right) = \frac{-1}{\rho}\frac{\alpha}{\alpha z}\left(\frac{\alpha P}{\alpha r}\right) - \frac{\mu}{\rho}\left(\frac{\alpha}{\alpha z}\left(\frac{\alpha^2 Vr}{\alpha z^2}\right) + \frac{\alpha}{\alpha z}\left(\frac{1}{r}\frac{\alpha Vr}{\alpha r}\right) + \frac{\alpha}{\alpha z}\left(\frac{\alpha^2 Vr}{\alpha r^2}\right) - \frac{\alpha}{\alpha z}\left(\frac{Vr}{r^2}\right)\right)$$

$$= \frac{-1}{\rho}\frac{\alpha}{\alpha z}\left(\frac{\alpha P}{\alpha r}\right) + \frac{\mu}{\rho}\left(\frac{\alpha^2}{\alpha z^2} + \frac{1}{r}\frac{\alpha}{\alpha r} + \frac{\alpha^2}{\alpha r^2} - \frac{1}{r^2}\right)\cdot\frac{\alpha Vr}{\alpha z} \tag{12}$$

Subtracting both sides of equations each other, we get

$$\left\{\frac{\alpha}{\alpha t} - \frac{\mu}{\rho}\left(\frac{\alpha^2}{\alpha z^2} + \frac{1}{r}\frac{\alpha}{\alpha r} + \frac{\alpha^2}{\alpha r^2} - \frac{1}{r^2}\right)\right\}\cdot\left(\frac{\alpha Vr}{\alpha z} - \frac{\alpha Vz}{\alpha r}\right) = 0 \tag{13}$$

By equ 7 and equ 8, then second factor in equ 13 is

$$\frac{\partial Vr}{\partial z} - \frac{\partial Vz}{\partial r} = \frac{1}{r}\left(\frac{\partial^2 \psi}{\partial z^2} - \frac{1}{r}\frac{\partial \psi}{\partial r} + \frac{\partial^2 \psi}{\partial r^2}\right) \tag{14}$$

Inputting equ 14 into equ 13, we obtain,

$$\left\{\frac{\partial}{\partial t} - \frac{\mu}{\rho}\left(\frac{\partial^2}{\partial z^2} + \frac{1}{r}\frac{\partial}{\partial r} + \frac{\partial^2}{\partial r^2} - \frac{1}{r^2}\right)\right\} \cdot \left\{\frac{1}{r}\left(\frac{\partial \psi^2}{\partial z^2} - \frac{1}{r}\frac{\partial \psi}{\partial r} + \frac{\partial^2 \psi}{\partial r^2}\right)\right\} = 0 \tag{15}$$

The second factor in equ 15 can be modified as following by recognizing the meaning of the differential operator (this step of the mathematical treatment is refered in the appendix)

$$\left\{\frac{\partial}{\partial t} - \frac{\mu}{\rho}\left(\frac{\partial^2}{\partial z^2} + \frac{1}{r}\frac{\partial}{\partial r} + \frac{\partial^2}{\partial r^2} - \frac{1}{r^2}\right)\right\} \cdot \left\{\frac{\partial^2}{\partial z^2} + \frac{1}{r}\frac{\partial}{\partial r} + \frac{\partial^2}{\partial r^2} - \frac{1}{r^2}\right\} \cdot \frac{\psi}{r} = 0 \tag{16}$$

Define the spatial differential operator as

$$D = \frac{\partial^2}{\partial z^2} + \frac{1}{r}\frac{\partial}{\partial r} + \frac{\partial^2}{\partial r^2} - \frac{1}{r^2} \tag{17}$$

Then we can get the simple operator equation.

$$\left(\frac{\partial}{\partial t} - \frac{\mu}{\rho} D\right) \cdot D \cdot \frac{\psi}{r} = 0 \tag{18}$$

Here we assume that eq 18 can be given by summing two independent solutions ψ_1 and ψ_2 as, $\psi = \psi_1 + \psi_2$.

Then either ψ_1 or ψ_2 must satisfy either of those equations.

$$D\left(\frac{\psi_1}{r}\right) = 0 \qquad \left(\frac{\partial}{\partial t} - \frac{\mu}{\rho} D\right) \cdot \frac{\psi_2}{r} = 0$$

Operating these differentiations by eq 17, next equations are induced.

$$\frac{\partial^2 \psi_1}{\partial z^2} - \frac{1}{r}\frac{\partial \psi_1}{\partial r} + \frac{\partial^2 \psi_1}{\partial r^2} = 0 \tag{19}$$

$$\frac{\partial^2 \psi_2}{\partial z^2} - \frac{1}{r}\frac{\partial \psi_2}{\partial r} + \frac{\partial^2 \psi_2}{\partial r^2} - \frac{\rho}{\mu}\frac{\partial \psi_2}{\partial t} = 0 \tag{20}$$

Summing equ 19 and equ 20 abd utilizing the relation $\psi = \psi_1 + \psi_2$, then

$$\frac{\alpha^2\psi}{\alpha z^2} - \frac{1}{r}\frac{\alpha\psi}{\alpha r} + \frac{\alpha^2\psi}{\alpha r^2} = \frac{\rho}{\mu}\frac{\alpha\psi_2}{\alpha t} \tag{21}$$

Inputting equ 21 into equ 9 and equ 10

$$-\frac{1}{r}\frac{\alpha^2\psi}{\alpha r \alpha t} - \frac{\mu}{\rho}\frac{1}{r}\frac{\alpha}{\alpha r}\left(-\frac{\rho}{\mu}\frac{\alpha\psi_2}{\alpha t}\right) = -\frac{1}{\rho}\frac{\alpha P}{\alpha z} \tag{22}$$

$$\frac{1}{r}\frac{\alpha^2\psi}{\alpha z \alpha t} - \frac{\mu}{\rho}\frac{1}{r}\frac{\alpha}{\alpha z}\left(\frac{\rho}{\mu}\frac{\alpha\psi_2}{\alpha t}\right) = -\frac{1}{\rho}\frac{\alpha P}{\alpha r} \tag{23}$$

Because of $\psi_1 = \psi - \psi_2$, then equ 22 and equ 23 are

$$\frac{\rho}{r}\frac{\alpha^2\psi_1}{\alpha r \alpha t} = \frac{\alpha P}{\alpha z} \tag{24}$$

$$-\frac{\rho}{r}\frac{\alpha^2\psi_1}{\alpha z \alpha t} = \frac{\alpha P}{\alpha r} \tag{25}$$

To eliminate ψ_1, multiplying r both equations in each sides and differentiate equ 24 with respect to r and defferentiate equ 25 with respect to z, then

$$\frac{\alpha}{\alpha z}\left(r\frac{\alpha P}{\alpha z}\right) + \frac{\alpha}{\alpha r}\left(r\frac{\alpha P}{\alpha r}\right) = 0$$

Therefore the partial defferential equation which include only function P is

$$\frac{\alpha^2 P}{\alpha z^2} + \frac{1}{r}\frac{\alpha P}{\alpha r} + \frac{\alpha^2 P}{\alpha r^2} = 0 \tag{26}$$

B. The solution of the equation which satisfy P.

As P is the cyclic function which depends on the cardiac rhythm, we assume the linear cyclic solution as in the following form,

$$P = P(r) * \exp\{in * (t - z/c)\} \tag{27}$$

n : the angular velocity $n = 2\pi f$

c : the pulse wave velocity

i : imaginary unit.

Which means one can separate the solution into time and space domaine and these quantities should have no interaction.

Inputting equ 27 into equ 26, equ 26 is reduced to the 0 order bessel type differential equation of P (r).

$$\frac{d^2P(r)}{dr^2} + \frac{1}{r}\frac{dP(r)}{dr} - \frac{n^2}{c^2}P(r) = 0 \tag{28}$$

As the limitting case r-0, there exists a finite solution. So the second order solution should be discarded. Then one can easily obtain the solution as

$$P(r) = J(i*n*r/c)$$

Therefore P can be given in the form as,

$$P = A_1 J_0(inc/r)*\exp(i*n*(t-z/c)) \tag{29}$$

c) The solutions of the linearlized Navier-Stokes equations

Assuming the linearity of the arterial system, then the frequency of the input and output must be idential. Consequently the blood flow velocities Vz, Vr can be written in the form simmilary as input pressure,

$$Vz = w(r)*\exp\{i*n*(t-z/c)\} \tag{30}$$

$$Vr = v(r)*\exp\{i*n*(t-z/c)\} \tag{31}$$

$$P = P(r)*\exp\{i*n*(t-z/c)\}$$

Inputting equ 27, equ 30, equ 31 into equ 5, 6, then

$$inW = \frac{1}{\rho}\frac{in}{C}P(r) + \frac{\mu}{\rho}\left(\frac{\alpha^2 V}{\alpha r^2} + \frac{1}{r}\frac{\alpha W}{\alpha r} + \left(\frac{-in}{C}\right)^2 W\right) \tag{32}$$

$$inV = \frac{-1}{\rho}\frac{\alpha P(r)}{\alpha r} + \frac{\mu}{\rho}\left(\frac{\alpha^2 V}{\alpha r^2} + \frac{1}{r}\frac{\alpha V}{\alpha r} + \left(\frac{-in}{C}\right)^2 V - \frac{V}{r^2}\right) \tag{33}$$

Which is the equations including variable r only.

The continuity equation 4 also should be modified into following form

$$\frac{\alpha V}{\alpha r} + \frac{V}{r} + W\frac{-in}{C} = 0 \tag{34}$$

Rearranging equ 32, then

$$\frac{\alpha^2 W}{\alpha r^2} + \frac{1}{r}\frac{\alpha W}{\alpha r} - \left\{\frac{\rho in}{\mu} - \left(\frac{-in}{C}\right)^2\right\} W = \frac{1}{\rho}\frac{\rho}{\mu}\left(\frac{-in}{C}\right) P(r) \tag{35}$$

Which is the 0th order Bessel type differential equation. In general about for the Bessel type equation as

$$Y'' + Y'/z - (\beta^2 + \nu^2/z^2)\, Y = 0$$

The finite converging solution is $Y = J\nu(i\beta z)$

Therefore in equation 35, putting

$$\beta^2 = \frac{in\rho}{\mu} + \frac{n^2}{C^2} \tag{36}$$

Then the solution of the equ 31 is

$W_1 = C1\, J_0(i\beta r)$

C1 : integral coefficient

on the other hand the specific solution is

$$-\beta^2 W_2 = \frac{1}{\rho}\frac{\rho}{\mu}\left(\frac{-in}{C}\right) P(r)$$

Therefore utilizing equ 29,

$$W^2 = \frac{in}{\beta^2 \mu C} P(r) = \frac{inD\, J_0(inr/C)}{\beta^2\, \mu\, C} \tag{37}$$

Then the general solution is

$$W = C_1 J_0(i\beta r) + \frac{inD\, J_0(inr/C)}{\beta^2\, \mu\, C} \tag{38}$$

As for the radial direction V, simillary rearranging the equ 33

$$\frac{\alpha^2 V}{\alpha r^2} + \frac{1}{r}\frac{\alpha V}{\alpha r} - \left(\frac{\rho\, in}{\mu} - \left(\frac{-in}{C} \right)^2 + \frac{1}{r^2} \right) V = \frac{1}{\rho}\frac{\rho}{\mu}\frac{\alpha P(r)}{\alpha r} \tag{39}$$

Utilizing equ 36, equ 39 is converted into 1st order bessel type differential equation. The solution of it is given as

$$V_1(r) = C_2 J_1(i\beta r) \tag{40}$$

C2 : integral coefficient.

As for the $P(r) = D * J_0(i * n * r/c)$, (here the integral coefficient is redifined as D instead of A1). According to the formula about the differentiation of the Bessel function,

$$\frac{dZ_\nu(r)}{dr} = \frac{\nu}{r} Z_\nu(r) - Z_{\nu+1}(r)$$

Then,

$$\frac{dP(r)}{dr} = -D\frac{in}{C} J_1(inr/C)$$

Therefore the specific solution is given as

$$V_2 = D\frac{in}{C}\frac{J_1(inr/C)}{\mu\beta^2} \tag{41}$$

Then the general solution is

$$V = C_2 J_1(i\beta r) + \frac{Din}{C}\frac{J_1(inr/C)}{\mu\beta^2} \tag{42}$$

Now we set.

$$\alpha_0^2 = \frac{i^3 a^2 n}{\nu} = i^3 \alpha^2 \qquad : \nu = \frac{\mu}{\rho} \tag{43}$$

$$\beta_0 = \frac{ina}{C} \tag{44}$$

α : the Womersleys coefficient.

a : the internal radius of the artery.

since $n^2/C \to 0$, then $\beta^2 = \frac{in\rho}{\mu}$ (45)

Then

$$\frac{Din}{C\mu\beta^2} = -\frac{D\beta_0 a}{\mu\alpha_0^2} \tag{46}$$

$$i\beta = \left(\frac{i^3 n\rho}{\mu} + \frac{i^3 n^2}{C^2}\right)^{1/2} = \left(\frac{\alpha_0^2}{a^2} + \frac{\beta_0^2}{a^2}\right)^{1/2}$$

(the pulse wave velocity C is 13-18m/sec in human arterial system).

By utilizing the α_0 and β_0 , changing the integral costants C1, C2, D into A1, A2, the solution 38, 42 are expressed in the following

$$W = Vz = A_2 \frac{(\alpha_0^2 + \beta_0^2)^{1/2}}{\alpha_0 J_0(\alpha_0)} J_0\left(\frac{(\alpha_0^2 + \beta_0^2)^{1/2} r}{a}\right) - \frac{A_1 \beta_0 a}{\mu \alpha_0^2} * J_0\left(\frac{\beta_0 r}{a}\right)$$

$$* \exp\{in(t - z/c)\} \tag{48}$$

$$Vr = A_2 \frac{\beta_0}{\alpha_0 J_0(\alpha_0)} J_1\left(\frac{(\alpha_0^2 + \beta_0^2)^{1/2} r}{a}\right) - \frac{A_1 \rho \alpha}{\mu \alpha_0^2} J_1\left(\beta_0 \frac{r}{a}\right) * \exp\{in(t - z/c)\} \tag{49}$$

$$P = A_1 J_0\left(\beta \frac{r}{a}\right) * \exp\{in(t - z/c)\}$$

Now the coefficients are normalized by $\alpha\ J_0(\alpha_0)$.

Since $n/c \to 0$, so by equ 43 and equ 44, then $\alpha_0^2 + \beta_0^2 \to \alpha_0^2$

Putting $r/a = r/R = y$ and $i * n * a/c = k$, then we get the following form of solutions.

$$Vz = A_2 \frac{J_0(\alpha_0 y)}{J_0(\alpha_0)} - \frac{A_1}{\mu} \frac{ina}{C} \frac{a}{i^3 \alpha^2} J_0(ky) \tag{51}$$

$$Vr = A_2 \frac{\beta_0}{\alpha_0} \frac{J_1(\alpha_0 y)}{J_0(\alpha_0)} - \frac{\alpha}{\mu} \frac{kA_1}{i^3 \alpha^2} J_1(ky) \tag{52}$$

If one redifines $A_2\ \beta_0/\alpha_0=C_2$, this form is idential with what Womersley had induced.

D. The satisfactory condition for the conservative law.

As those solutions and equations are based on the Stokes stream function, as mentioned in eq 7, 8 which had been assumpted to satisfy the conservative law eq 4, eq 51 and 53 should be examined whether the continuity equation 4 satisfy. The continuity equation should be converted into

$$\frac{1}{y}\frac{d(V\ r\ y)}{dy}=\frac{inR}{C}Vz \tag{53}$$

Utilizing the differentiation formula of the Bessel function for equ 52

$$\frac{dJ_1(\alpha_0 y)}{dy}=J_0(\alpha_0 y)-\frac{J_1(\alpha_0 y)}{\alpha_0 y}\alpha_0$$

We now put

$$\frac{A_2\ \beta_0}{\alpha_0}=C_2$$

Then the left side of the equ 53 becomes

$$\frac{C_2}{J_0(\alpha_0)}\left\{\alpha_0 J_0(\alpha_0 y)-\frac{J_1(\alpha_0 y)}{y}\right\}-\frac{RkA_1}{\mu i^3\alpha^2}\left\{k\,J_0(ky)-\frac{J_1(ky)}{y}\right\}+C_2\frac{J_1(\alpha_0 y)}{J_0(\alpha_0)\,y}$$

$$-\frac{RkA_1 J_1(ky)}{\mu i^3\alpha^2 y}=C_2\frac{\alpha_0 J_0(\alpha_0 y)}{J_0(\alpha_0)}-\frac{Rk^2A_1}{\mu i^3\alpha^2}J_0(ky) \tag{54}$$

and the right side of equ 53 reduces to

$$\frac{inR}{C}A_2\frac{J_0(\alpha_0 y)}{J_0(\alpha_0)}-\frac{inR}{C}\frac{inR^2}{\mu C i^3\alpha^2}A_1 J_0(ky)$$

The equation 54 and and 55 should coincide. Therefore the coefficients of $J_0(\alpha_0 y)$, $J_0(k*y)$ in both side should be idential. Then

$$\frac{C_2}{A_2} = \frac{inR}{C\alpha_0} = \frac{inR}{C\alpha i^{3/2}}, \quad k = \frac{inR}{C} \tag{56}$$

This is exactly what we have put $k = i*n*a/c$. Therefore equ 51 and equ 52 satisfy the continuity equation 4.

Assuming $n/c \rightarrow 0$, the approximation formula for the Bessel function reduce the term J_0 ($i*n*R/c$) approaches to 1 and J_1 ($n*R*y/c$) reduces to $n*R*y/2c$.

Ultimately the blood flow velocities are given as followings

$$Vz = A_2 \frac{J_0(\alpha_0 y)}{J_0(\alpha_0)} + \frac{A_1}{\rho C} \tag{57}$$

$$Vr = \frac{inR}{2C}\left(A_2 \frac{2J_1(\alpha_0 y)}{\alpha_0 J_0(\alpha_0)} + y\frac{A_1}{\rho C}\right) \tag{58}$$

α : the Womersleys coefficient

E. Another solution of the blood flow velocity by the Stokes stream function.

In equ 19 and equ 20, assuming that the two stream function ψ_1 and ψ_2 is separable in time and space domaine, one can express the solution in the linear form as

$$\psi_1 : F(r)*\exp\{i*n*(t-z/c)\} \tag{59}$$

$$\psi_2 : G(r)*\exp\{i*n*(t-z/c)\} \tag{60}$$

Inserting equ 59 and equ 60 into equ 19 and equ 20,

$$\frac{d^2F}{dr^2} - \frac{1}{r}\frac{dF}{dr} - \frac{n^2}{C^2}F = 0 \tag{61}$$

$$\frac{d^2G}{dr^2} - \frac{1}{r}\frac{dG}{dr} - \frac{n^2}{C^2}G - \frac{in\rho}{\mu}G = 0 \tag{62}$$

Putting $F = r*f(r)$ and $G = r*g(r)$ and simple calculation brings us to the following Bessel type differential equations.

$$\frac{d^2f}{dr^2} + \frac{1}{r}\frac{df}{dr} + \left(-\frac{n^2}{c^2} - \frac{1}{r^2}\right)f = 0 \tag{63}$$

$$\frac{d^2 g}{dr^2} + \frac{1}{r}\frac{dg}{dr} + \left(- \frac{n^2}{C^2} - \frac{1}{r^2} - \frac{in\rho}{\mu} \right) g = 0 \tag{64}$$

Then putting as before equ 43 and equ 44 as

$$\beta = \frac{in}{C}, \quad \alpha = \left(\frac{n^2}{C^2} + \frac{in\rho}{\mu} \right)^{1/2}$$

Here we newly defined α as above and is different from the Womersley coefficient.
Then the solution can be easily attained as

$$f(r) = \frac{F}{r} = A J_1 (i\beta r) = A J_1 \left(\frac{inr}{C} \right)$$

$$g(r) = \frac{G}{r} = B J_1 (i\alpha r) = B J_1 \left(i \left[\frac{n^2}{C^2} + \frac{in\rho}{\mu} \right]^{1/2} r \right)$$

The J (i * x) is the pure imaginary number, then to have non trivial solution, the coefficiens should redifined as

$$A = A * i \qquad B = B * i$$

Then the Stokess stream function is given as following

$$\begin{aligned} \psi &= \psi_1 + \psi_2 = (F(r) + G(r)) * \exp\{i * n * (t - z/c)\} \\ &= r (A_1 J_1 (i\beta r) + \beta_1 J_1 (i\alpha r)) * \exp\{in (t - z/c)\} \end{aligned}$$

Because of the definition of the equ 7,

$$Vz = - \frac{1}{r}\frac{\alpha}{\alpha r} \left(r\, i (A J_1 (i\beta r) + B J_1 (i\alpha r)) \right) \tag{65}$$

Utilizing the differential equation of the 1st order Bessel function

$$\frac{d J_1 (i\beta r)}{dr} = \frac{d J_1 (i\beta r)}{d (i\beta r)} \frac{d (i\beta r)}{dr} = \left(J_0 (i\beta r) - \frac{J_1 (i\beta r)}{i\beta r} \right) i\beta$$

Then

$$Vz = \frac{-i}{r}\left(A J_1(i\beta r) + \beta J_1(i\alpha r) + r\left(A\left(J_0(i\beta r) - \frac{J_1(i\beta r)}{i\beta r}\right) i\beta \right.\right.$$

$$\left.\left. + B\left(J_0(i\alpha r) - \frac{J_1(i\alpha r)}{i\alpha r}\right) i\alpha \right)\right)$$

$$= \frac{-i}{r}\left(r\, i\beta A J_0(i\beta r) + r\, i\alpha B J_0(i\alpha r)\right)$$

$$= A\beta J_0(i\beta r) + B\alpha J_0\left(i\left(\frac{n^2}{C^2} + \frac{in\rho}{\mu}\right)^{1/2} r\right) \qquad (66)$$

As for the radial velocity only, the differentiation with respect to z reduces to

$$Vr = A\frac{n}{C} J_1(i\beta r) + B\frac{n}{C} J_1(i\alpha r) \qquad (67)$$

As C (pulse wave transmission velocity) is 13-18m/sec in human arterial system, then $n/c \to 0$, and $J_0(i\beta r) \to 1$

$$\alpha = i^{1/2}\left(\frac{n\rho}{\mu}\right)^{1/2} = i^{1/2} * \alpha\bigstar$$

$$Vz = \frac{iAn}{C} + B\, i^{1/2}\, \alpha\bigstar J_0(i^{3/2}\alpha\bigstar r)$$

At the innersurface of the arterial wall, the blood flow velocity attains the finite value

$Vz(r=R) = V\bigstar$, Then

$$V\bigstar = \frac{Ani}{C} + B\, i^{1/2}\, \alpha\bigstar J_0(i^{3/2}\alpha\bigstar R)$$

So the coefficient B is

$$B\, i^{1/2}\alpha\bigstar = \frac{V\bigstar - A\, n\, i/C}{J_0(i^{3/2}\alpha\bigstar R)}$$

Therefore

$$Vz = \frac{A\,n\,i}{C} + \left(V\bigstar - \frac{Ani}{C} \right) \frac{J_0 (i^{3/2} \alpha \bigstar r)}{J_0 (i^{3/2} \alpha \bigstar R)}$$

Here redefine the coefficient and parameter as following

$$\frac{Ain}{C} = \frac{A_1}{\rho C}, \quad \left(V\bigstar - \frac{Ani}{C} \right) = C_1$$

$$R * \alpha\bigstar = \alpha \ , y = r / R$$

Here the coefficient α idential with womersleys coefficient.

Then the longitudinal blood flow velocity is obtained as

$$Vz = \frac{A_1}{\rho C} + C_1 \frac{J_0 (\alpha\, i^{3/2}\, y)}{J_0 (\alpha\, i^{3/2})} \tag{68}$$

The same procedure bring us to the following equation with respect to the radial blood flow velocity is

$$Vr = \frac{inR}{2C} \left(C_1 \frac{2 J_1 (\alpha\, i^{3/2}\, y)}{\alpha\, i^{3/2} J_0 (\alpha\, i^{3/2})} + y \frac{A_1}{\rho C} \right) \tag{69}$$

The eq 68 and 69 are idential with eq 57 and eq 58 respectively.

Therefore the solutions of the linearlyzed Navier-Stokes equation are obtained in the form as eq 57, 58 or eq 68, 69.

APPENDIX

$$\left(\frac{\alpha^2}{\alpha z^2} + \frac{1}{r} \frac{\alpha}{\alpha r} + \frac{\alpha^2}{\alpha r^2} - \frac{1}{r^2} \right) \left(\frac{\psi}{r} \right)$$

$$= \frac{1}{r} \frac{\alpha^2 \psi}{\alpha z^2} + \frac{1}{r} \left(- \frac{\psi}{r^2} + \frac{1}{r} \frac{\alpha \psi}{\alpha r} \right) + \frac{\alpha^2}{\alpha r^2} \frac{\psi}{r} - \frac{\psi}{r^3}$$

$$= \frac{1}{r}\frac{\alpha^2\psi}{\alpha z^2} + \frac{1}{r^2}\frac{\alpha\psi}{\alpha r} - \frac{\Psi}{r^3} + \frac{\alpha}{\alpha r}\left(\frac{1}{r}\frac{\alpha\psi}{\alpha r} - \frac{\psi}{r^2}\right) - \frac{\psi}{r^3}$$

$$= \frac{1}{r}\frac{\alpha^2\psi}{\alpha z^2} + \frac{1}{r^2}\frac{\alpha\psi}{\alpha r} - \frac{2\psi}{r^3} - \frac{1}{r^2}\frac{\alpha\psi}{\alpha r} + \frac{1}{r}\frac{\alpha^2\psi}{\alpha r^2} - \frac{1}{r^2}\frac{\alpha^2\psi}{\alpha r} + \frac{2\psi}{r^3} .$$

$$= \frac{1}{r}\frac{\alpha^2\psi}{\alpha z^2} + \frac{1}{r}\frac{\alpha^2\psi}{\alpha r^2} - \frac{1}{r^2}\frac{\alpha\psi}{\alpha r}$$

$$= \frac{1}{r}\left(\frac{\alpha^2\psi}{\alpha z^2} - \frac{1}{r}\frac{\alpha\psi}{\alpha r} + \frac{\alpha^2\psi}{\alpha r^2}\right)$$

DISCUSSION

In the first chapter of the series of mathematical modeling of the cardiovascular system, we have developed a distributed parameter model of human arterial system. This papre treated with the mathematical expansion for the pure blood flow velocities which does not include the arterial wall properties. To obtain the velocities (which are the solutions of the Navier-Stokes equations), we made some assumptions about the blood and artery. We discuss firstly the significance of modeling and the Navier-Stokes equation, then expand the discussion mainly about the blood properties.

I. The modeling of the biological system.

There are several candidates of the models which can express the cardiovascular system. To describe the effects of the cardiovascular elements on the blood flow two typical models exist.

First is the lumped circuit model in which many biophysical properties of the arterial wall and blood are gathered together.

The classical but representative model is the windkessel type model as Frank had suggested. Such model is composed of the reistance and compliance only. So each character of the element which compose the arterial blood flow and the effects on the flow wave were made obscure. Furthermore it can be seen which component (for example whether arterial wall thinkness or the blood density) mainly contribute to the change of the total arterial resistance or the arterial compliance. Of course such lumped circuit model cannt represent the transmission phenomenone even much elements are incorporated, since this type of model never contains the variable x. However to look the dynamical system macroscopically and analyze overall behaviour of the large system, the

lumped circuit model is suitable.

On the other hand the distributed parameter model is arranged utilyzing R,L,C,G in the axial direction and radial direction (the ladder circuit) depending on the distance from the entry zone. So a simple increment of the number of the elements brings us to more precise representation of the transmission phenomenone of flow wave. But when one should look the cardiovascular system exclusively and to analyze the effects of the change of some compartment of the artery (such as aortic arch or small arterioles), one would confuse to treate such distributed model since the parameters all change continuously in the special domain and these sequential parameter change may obscure the segmental change which one concerns.

In any way the selection of the model is depend on the purpose or phenomenone that one wish to analyze and to disclose. In the first series of the papers we analyze the pulse wave transmission phenomenone. So we adopted the distributed parameter model.

The candidates of the arterial that satisfy these mentioned assumptions are rather many in human arterial system. We apply this model for the middle sized arterial system such as femoral artery or brachial artery.

II. The solution of the Navier-Stokes equation.

We have deduced the blood flow velocities in the longitudinal and radial directions from linearlyzed Navier-Stokes equations. As a conventional way, we utilyzed the Stokes stream function and induced the differential equation about the input pressure P. The pressure was shown to be a Bessel type function.

There is at least two process to obtain the blood flow velocity from the N-S equations utilyzing the Bessel type function of blood pressure P. First is to solve the linearlyzed N-S equation as a Bessel type 0th order or 1st order differential equation. The second is from differentiating the Stokes stream function. In either solution process, the Bessel function and its differential are included. By setting adequate coefficients, these solutions proved to coinside each other and also satisfy the conservative law.

The linearlyzed solutions include those parameters as $y=r/R$, ρ (the blood density), C (the pulse wave velocity), α (the Womersley coefficient : $r * \sqrt{n * \rho / \mu}$, n : the angular velocity). The Womersleys coefficient is the ratio of radius of tube and thinkness of the vibratory boundary layer and this ratio directly relates to the velocity profiles of the flow in an artery especially for the pulsatile flow.

Therefore the blood velocity are the function of both the blood and arterial wall properties. This interrelationships intimately correlate with the specificity of the distributed parameter model and

transmission line theory.

III. The signifcance of the Navier-stokes equation

When the fluid moves, it travels continuously in time and space domain. Therefore it is the velocity in the time and space domain and not position that should satisfy the N-S equatuion. In general the N-S equation can be expressed as following form,

$$DV/Dt = F - \text{grad } P + f.$$

D/Dt is the lagurange's differential operator and indicates the change of the velocity of each particle in the fluid. This also corresponds to the inertial force that act on the unit mass of the fluid. F is the external force. Grad P is the pressure gradient. f is the viscous stress inherent of the viscous fluid. The N-S equation represent the balancing state of the forces acting on the viscous fluid. As in the analysis of the electromagnetic field, the interruption of these equation is different when the fluid dynamical phenomenone is looked in a macroscopic view or microscopic view. Applying the conservative law of the momentum to a given time and space domain in the arbitrary point of the fluid and squizing these region infinitely small, then one can obtain the limitting equation. That is the Navier-Stokes equation. Therefore it is suitable to express minuetly the local flow state qualitatively. On the other hand to apply the conservative law to a finite time and space domain, the N-S equation should be integrated. This is the integral equation of the momentum and applied to the analysis of the macroscopic flow state. In such a stand point N-S equation also can represent the turbulent flow and movement of the compressible fluid. The N-S equation is different in form of the solution depending on the character and quality of the flow which the N-S equation express. In the case of the Reynolds number smaller than 1, the dominative term is the dissipative term and the solutions are always stable. However when Re increase, the convective acceleration term increase and the differentiation of the flow should occurs. Such that the main flow part is the one viscous flow and in the boundary layer, the shearing flow exist. Until the Re exceed 1000, the flow is mainly laminar but for the larger Re, there develops the turbulent flow and the analysis is extremely difficult. About for the practical solution of the N-S equation, many approaches have been presented such as

1. Convertion into the difference equations
2. The utilization of the functional derivation of the Navier-Stokes equation and apply the variational princple.
3. The linearization of the Navier-Stokes equation.

In our studies based on the linearization of the whole system we adoppted the 3rd analyzing method. The reasons for the validity of utilyzation of the linearity are discussed in the 5th papaer

of our studies.

IV. The non Newtonian property of the blood.

The fluid which obeys the law of the Newton is called the Newtonian fluid. In the newtonian fluid the flow velocity gradient D paralells with the shearing stress (S). Then the relation $D=1/\eta * S$ holds. The proportion coefficient η is the viscos coefficient. Such law stands only for the simple liquid and gas. For the deformative substance, another expression of the formula is $\gamma = S/n$, where γ is the shear rate. As a result the shear rate parallels with the shear stress. However in the fluid such as colloid solution, a simple parallel relation does not hold between the shear rate and the shear stress. In the non Newtonian fluid, the viscosity of the fluid is defined as $\eta a = S/\gamma$ which is the apparent viscosity. In the blood the apparent viscosity decrease with increase of γ and this phenomenone is called the shear thinning.

The apparent viscosity depend on the following factors.

a. The temperature of the liquid.

b. The length of the tube.

c. The diameter of the tube.

d. The concentration of the RBC and Haematocrit.

e. The velocity of the blood flow (the shear rate)

a) The apparent viscosity falls with the increase of the temperature. But except for the extermely low shear rate (below 1/sec), the viscosity is almost constant as 10-40c

b) The viscosity changes depending on the length of the tube which character is called the thixotropic effect. This effect can be neglected since the length of our model will be assumed to 60 cm and the radius of the artery 0.35cm at most. So the length of the artery is 100 times larger than its diameter.

c) When the diameter of the tube decreased below hundred μ m, the apparent viscosity decreased with the decrease of the tube diameter. This phenomenone is called the Fahraeus-Lindquvis effect. This phenomenone is explained by the local change of the Haematcrit (the axial accumulation) and is negligible for the range of radius as femoral artery.

d) The apparent viscosity increase parallely with Hct increase, yet the viscosity striongly depends on the diameter of the viscometer. According to Whittaker and Winton (1933) such results are also obtained in Vivo (the perfused canine hind limb). Their data were clearly coincided with data obtained by the viscometer when the results are corrected by the inertial losses [7].

e) The relation between the shear stress applied on the blood and resulting shear rate is expressed in the Casson's equation

$s = k * \gamma + C$ s : shear stress, γ : shear rate.

C is the shearing stress neccesary for begining the fluid movement and is called the yield stress. As a matter of course the viscosity coefficient also depends on the Hct and dereases with the increase of the shear rate. However this tendency is explicit only for the shear rate smaller than 10/sec. and is constant for the range greater than 100/sec. at any Hct value [8]. However these results are obtained under the condition of steady flow and in small diameter glass tube. Nevertheless in human arterial system, the shear rate for the ascending aorta is 190/sec at wall (mean 130/sec) and for the large artery such as the femoral artery, the shear rate is 700/sec at wall (mean 470/sec) [9]. Therefore theoretically the viscos coefficient shoud be independent of the shear rate. If this holds ture, then the relation between the pressure difference P and the flow rate should parallel under the condition of the steady flow. However in general these relation is all nonlinear. Bayliss [10] firstly measured this relation at Hct = 49% for the steady flow using the tube with the radius 408 μm, length 155cm. The resulting pressure flow relation was nonlinear. So the Poisseulie's law does not hold.

The conditions that hold for the Poisseuile law originate mainly in the Newtonian properties of the blood. These are followings.

1. The tangential stress between the shearing cylindrical laminar of the fluid parallel with the velocity gradient across the laminae. This indicate the consistency of the proportionality of the viscous coefficient and is independent of the velocity gradient (the shear rate).

2. The flow is laminar which means the viscous forces responsible for energy dissipation are parallel to the axis of the tube.

3. The velocity profile should be parabolic.

4. The fluid is homogeneous.

These conditions are demanded however only in the steady flow. In the non Newtonian fluid such as the blood, some different experimental results were reported. Rivlin (1948) already had shown the existence of the normal components of the stress tensor in the non Newtonian fluid.

Haynes and Burton (1955) [11] showed the existence of the dissipative normal forces to the axis of the tube and concluded the blood flow was not laminar. They also calculated the viscous coefficient along the radial direction. It increased from 4P at the wall to 6P at the axis by the axial accumulation. After these study they analyzed the effects of the non-Newtonian properties of the blood on the pressure-flow relation in the glass tube of the radius from 50 to 800 μm. The experimentally obtained curves had become linear as the flow rate increased. Based on these data, curves of shear rate as the wall 4Q/R3 versus shearing stress at the wall PR/2 were constructed

under the steady flow. These curves were all linear at shear rate PR/2 greater than 20 dyn/cm. In the human artery, the stress at the wall is 60 dyn/cm for large artery (R = 0.5cm) and 93 dyn/cm for the terminal artery. So theoretically in the femoral artery, obviously PR/2 $>$ 20 dyn/cm. Then the relation should be linear and the non-Newtonian properites should not be reflected. Since these results were obtained under the condition of the steay flow in the small diameter, and in the rigid circular tube and above mentioned data were obtained all in Vitro and some differences may exist between the results obtained in Vivo.

Benis [7] and his colleges assesed the effects of these non Newtonian properties of the blood on the non linear relation of the pressure-flow. These experiments were performed in the perfused isolated hind paw of the dog. They used the specific parameter Rv/Rvo (Rv : the viscous vascular resistance, Rvo : viscous flow resistance for cell free perfusate). This variable was not affected by the vascular geometric parameter and has been corrected for the inertial losses. So it reflected only the rheological behaviour of the perfusate. The change of the Rv/Rvo could be regarded to originate in the non Newtonian viscosity of the RBC suspension. The Rv/Rvo increased with the increase of Hct and with decrease of the normaliyzed flow rate. At Hct of 20.5%, the increase of the relative normalyzed flow rate of about 240% reduced the Rv/Rvo only for 8%. For the case of Hct = 50.6%, the increase of relative normalized flow rate of 260% reduced the Rv/Rvo only for 11%. Consequently the significant change of the true viscosity occured only in the case of extremely large change of the flow rate. These observations were done under the perfused steady flow state and is not directly comparable with the pulsatile flow. Nevertheless even under the pulsatile flow such large flow rate change would not occur. So the viscosity should not change and can be regarded as independent of the flow rate.

V. The turbulence

For the steady state flow in the straight circular cylindrical tube, it has been known that the laminar flow transients to the turbulent flow if the conditions that satisfy the critical Reynolds number have been reached. The critical Renolds number is defined as

$Re = U * D * \rho / \mu$ (U: the blood flow velocity, D: the diameter of the tube, ρ: the blood density, μ: the blood viscosity)

According to the physical experiment the critical Reynolds number Re is calculated to be 2000 when the steady flow pass through the circular rigid tube. Calculating the Re for the case of human femoral artery, assuming Vz = 100cm/sec, D = 0.5cm, $\rho = 1.05$, $\mu = 0.03$, then Re = 1758 which is understimated. On the other hand by Whitmore (1968) [9], it was revealed that for the human ascending aorta, the Reynolds number ranged as $3600 < Re < 5800$, for the descending aor-

ta it ranged as 1200 ‹ Re ‹ 1500, for the large artery such as the diameter 0.2 ‹ Dcm ‹ 0.6, Re= 850. However these calculations were done on the assumption of the steady flow in the rigid circular tube. Practically the blood flow in the main arteries are pulsatile nature and the tube has enevitable visco-elastic properties. Consequently these simple mathematical theory can't be applicable. Some physical engineering experiments have been reported especially with respect to the pulsatile flow of the arterial system.

Sarpkaya (1967) [12] studied the conditions of the critical Reynolds number under the condition of the pulsatiling flow in the rigid tube. The Re was a function not only of the frequency parameter α (the Womersley's parameter) but also of the flow amplitude ratio lambda (the ratio of the periodic mean velocity versus total mean velocity). According to his data the critical Re was not a simple increasing function. It increased with lambda (the pulsatile flow velocity component) and had reached maximum value, then decreased exponentially. For example in the case of $\alpha = 4.0$, Re increased with lambda and had attained max=5150 for lambda=0.625, then it decreased to Re= 400 for lambda=1.0. For the case of $\alpha = 7.2$, Re attained max=2950 at lambda=0.28, then decreased to 0 at lambda=0.65. The Re increased with lambda and decreased with α. He concluded that for the same mean pressure gradient, the Re for the pulsatile flow is higher than steady flow. In addition the Re for the nonharmonic oscillation were lower than those of the harmonic pulsatiling flow. Nevertheless his data refered only to the ratio of lambda untill 0.95. These flow were mainly steady flow and the proprotion of the pulsatile components in his experiment was extremely small comparing with the actual flow. For example for the human aorta the flow ratio is 2 to 5 and for the femoral artery the ratio is 7 to 8. As the Re had decreased exponentially after attaining the max value, the large lambda would cause the Re decresed markedly for the large value of the frequency parameter α. So their results are not easily applicable for our studies.

Hino (1978) [13] analyzed the behaviour of the critical Re under the condition of the purely oscillatory pipe flow. So the flow ratio lambda was infinite. They used the following parameters Re, Rd ($= U * d/v$, $d = 2v/w$, v: the kinematic viscousity, w: the angular velocity) which is the Reynolds number defined in terms of U (flow velocity) and stokes layer thickness d. The relation of Re and Rd was Re = 2 * lambda * Rd. The lambda was the stokes parameter which relates the frequency parameter of Womersley as Re = 2 * lambda * Rd. Their data did not include the steady flow components. The types of the obserbed oscillatory flow could be classified into 4 types in terms of the Re and the stokes parameters. Each flow pattern depended on these parameters differentially. They were

1. The laminar flow

2. The distorted flow

3. The weakly turbulent flow

4. The conditional turbulence in which the turbulence appears only in the deccelerating phase of the flow although in the acceleration phase the flow transient to the laminar like flow. For the laminar or distorted laminar flow the critical Re increased with the decrease of lambda ($=1/2*\alpha$). For example when lambda$=4.5$, then Re$=670$ and lambda$=3.1$, then Re$=1330$. This pattern coinsided with the data of Sarpkaya. However these data were scattered and dissipated much and the interpretation was tentative. For the type 4 flow, the Re increased with lambda, for lambda$=1.45$ ($*2=\alpha$) then Re$=2900$ and for lambda$=3.85$, then Re$=4200$. In addition Hino's data was obtained under the condition of lucite circular pipes having the inner diameter of 14.5 to 29.7mm and the length of 400cm. These values were far from the physiological data of femoral artery.

Yellin (1967) [14] examined the laminar- turbulent transition process. They analyzed the factors that influence the transition from the laminar flow to turbulence under the condition of pulsetile flow. The growth rate of the turbulence decreased in the flow with low frequency and with large flow amplitude ratio which was the ratio between the steady component of the volume flow vs the amplitude of the periodic component of volume flow. He analyzed the effects of these parameters independently. Increasing the flow amplitude ratio from 0.1 to 0.3 as $\alpha=7$ (the frequency parameter of Womersley) had decreased the growth factor. decreasing the frequency parameter depressed the growth factor almost to 0 for the flow amplitude ratio of 0.33 (in this case the pulsatile component was rather small) at $\alpha < 7$ (Re$=2650$). This phenomenone was explicit in the lower Reynolds number. However for the frequency range of $0 < \alpha < 2.5$, the growth factor decreased with an increase of α. According to their data for the frequency range of $3 < \alpha < 8$, the remarkable decrease of the growth factor of the turbulence appeared. This tendency was reinforced much by only slight increase of the flow amplitude ratio which indicated the increase of the pulsatile component. In his studies he used the sinusoidal flow which differes from the practical arterial flow. So the results were not comletly comparable with the actual blood flow. However the fact the increase of the pulsatile component would decrease the turbulence gives much confidence of our study, for in the systolic phase of femoral artery, large acceleration induces a much greater flow and the flow amplitude ratio will be augmented to 6.5 to 8 times where even in his result shows much depression of the turbulence for the ratio of 0.1 to 0.3

About for the animal experiments firstly carried by Hele (1955) [15]. He had visualized the pulsatile nature of the blood using canine arterial system. He injected the dye in the canine femoral

artey in Vivo and analyzed the stream line by high speed cinematograpgy. As a result under the condition of the pulsatile flow, only laminar flow existed and the turbulence could n't be observed. On the other hand in the Rabitt abdominal aorta McDonald (1952) [16], discovered the existence of the turbulence. The difference between the results of Hale and McDonald would originate in the heart rate. The rabitt's HR was twice larger than that of the dog. This factor is comparable of the Sarpkaya's data. Dick [17] analyzed the relation ship between the turbulence and the power spectrum density of the nonlinear components that were included in the pressure flow relation in the canine aorta. By injecting the Norepinephrine, the nonlinear components of the power spectrum density were markedly decreased and the blood pressure- flow relation approached to the linear one. Nevertheless the turbulance did not changed. Based on these physical experiments, he infered the independency of turbulence and the nonlinear blood pressure-flow relation.

Associating these theoretical and experiments results, the turbulence can be infered to be small in pulsatile flow in the viscoelastic middle sized artery such as in the femoral artery.

Further more even if one assumes the existence of the turbulence, the effects of the turbulence on the linear or the nonlinear blood pressure-flow relation would be small in comparison with the other factors. Recently the analysis of the turbulence itself have been developed extensively and elaborously. But the mathematical and physical treatment is extremely difficult. One connot express the component of the turbulence and laminar flow in an idential equation.

VI. The internal radius.

In this paper the radius of the artery was assumed to be indepenent of the change of the pressure and the distance from the entry zone. However the radius is never constant during the cardiac cycle and along the given compartment of the arterial segment. The radius changes in time and space domain. These cubic deformation are transmitted along the arterial segments. Since the components of the arterial wall changes depending on the distance from the entry zone, there should developement of the shearing or bending stress in the arterial wall. Then these forces will make the deformation complex. As a matter of course such complicated wall deformation cause the change of the radius intricated. Consequently the radius of the artery is in itself the function of the pressure, distance and the stress. So one cannot treate the radius as a simple independent variable.

The relation between the radius and the pulsatile pressure had been already reported. Barnett (1961) [18] firstly measured the $\Delta R/Rs$ ($R=Rd$ (the diastolic radius)- Rs (the systolic radius)) of the descending aorta in the living dog. He reported that at the BP 60mmHg $<$ BPmmHg $<$ 140mmHg, it ranged as $2.5\% < \Delta R/rs < 7\%$. Furhermore the per unit pressure change (dynamical

extensibility index) $\Delta R/\Delta P$ was 0.022 to 0.048 cm/mmHg. In addition there existed close relation between the radius and pressure. The relative coefficient was $\gamma = 0.9$. Patel (1963,64) [19,20] analyzed further minutely for the human and canine arterial system in Vivo. For human ascending aorta., pulmonary artery, carotid artery and the femoral artery, the $\Delta R/Rs$ were 0.091, 0.107, 0.011, 0.013 respectively. The $\Delta R/\Delta P * 10$ cmH_2O were 1.8, 8.77, 0.071, 0.07 respectively. For canine ascending aorta, descending aorta, abdomainal aorta and the brachial artery, the $\Delta R/Rs$ were 0.0458, 0.035, 0.0075, 0.0198 respectively. The $\Delta R/\Delta P * 10$ cmH_2O were 1.472, 0.943, 0.126-0.211, 0.209 respectively. The higher values of the $\Delta R/Rs$ in the pulmonary artery were due to the proximity to the heart and the effect of the respiration.

Arndtt (1968) [21] also measured the change of the radius in the human carotid artery in Vivo. The results were $\Delta R/Rs = 0.0143$, $\Delta R/\Delta P * 10$ cmH2o $= 0.855$. The internal radius increased linearly with the blood pressure within the BP range of 60mmHg ‹ BPmmHg ‹ 130mmHg.

Cox,R,H (1975) [22] emphasized the dependency of the change of the radius on the pressure in the living dog. For descending aorta with 90mmHg ‹ BPmmHg ‹ 110mmHg, $\Delta R/Rs = 0.083$, $\Delta R/\Delta P = 0.63$cm/mmHg. For the abdominal aorta with 145mmHg ‹ BPmmHg ‹ 190mmHg, $\Delta R/Rs = 0.022$, $\Delta R/\Delta P = 0.456$cm/mmHg. For the subclavicular artery with 75mmHg ‹ BPmmHg ‹ 90mmHg, $\Delta R/Rs = 0.024$. For the carotid artery with 130mmHg ‹ BPmmHg ‹ 170mmHg, $\Delta R/Rs = 0.021$. For femoral artery with 120mmHg ‹ BPmmHg ‹ 180mmHg, $\Delta R/Rs = 0.023$, $\Delta R/\Delta P = 0.196$cm/mmHg.

Associating these results, the change of the radius due to the pulse pressure in the femoral artery is 2% to 3% at best which depend of course on the range of the change of the pressure. On the other hand data obtained in Vitro state are conflicted and many different results have been reported.

Tickner (1967) [23] presented that the results of the canine branchial artery under the strong longitudinal tethering with BP 75mmHg ‹ BPmmHg ‹ 300mmHg. The internal radius and the outer radius maintained almost constant value independent of the internal pressure change.

Cox (1975,76) [24,25] analyzed the canine iliac, carotid artery in Vitro. Untill BP 120mmHg, the internal radius showed nonlinear increase with pressure. Beyound this pressure, the radius did not change and kept a constant value even marked change occurs in the BP. Adding the Norepinephrine in this specimen and he activated the smooth muscle, then the behaviour of the radius changed from the previous result. Below BP = 83mmHg, the radius have constant value and was independent of pressure. With the range of 83mmHg ‹ BPmmHg ‹ 166mmHg, the radius changed parallely with the pressure. Over this pressure, the radius also maintained the constant value.

These response pattern was sigmoid.

Attinger (1966) [26] examined the change of radius by driving the wide range of pressure for canine arterial system in Vitro. He used the initial distending pressure about $2cmH_2O$ = 1.474mmHg. For the pressure range of 59mmHg < BPmmHg < 147.4mmHg, in the descending aorta the relative change of the radius was $1.45 < \Delta r/ro < 2.02$ and in the carotid artery the ratio was $1.83 < \Delta r/ro < 2.17$. He emphasized the marked nonlinearity of the radius and expressed the minimum change of the pressure induces a remarkable change in radius.

Associating these data, one cannot identify these results since different material, different measurement instrument and experimental conditions. However the difference between the data in Vivo and in Vitro seems to originate from the longitudinal tethering effect of the arterial wall in Vivo. Releasing these constraints would cause the specimen in free movable states. Consequently a minute change of the pressure makes the radius change surplyzingly. About for the impressive reports of Attinger, the distending pressure was markedly small. Naturally the % change of the radius increased much.

The mathematical model which include the change of radius had been reported only few cases. Womersley (1958) calculated the effects of change in the radius from R to $R+\xi$ (systolic to diastolic) on the mean blood flow velocity in the longitudinal direction w. He expanded the change of radius ξ with the help of the Fourier analysis and incorporated the Navier-Stokes equations. A much complex computations reduced that the effects due to the radius change contribute only 3.5% increment of the mean blood flow velocity. In addition this calculation had been done for the case of Co (the pulse wave velocity) was 500cm/sec and there was no longitudinal tethering (the free ending movement of the arterial wall). Therefore the value of the 3.5% should be regarded as an over estimated one. Based on another mathematical expansion he induced relation of the change of the radius and the blood flow velocity as $2\xi/R = w/C$ from the continuity equation. If one put w = 70cm/sec and C = 120cm/sec, then we get $\xi/R = 0.029$. That is almost identical value with the result of the animal experiment in Vivo. Therefore both in the mathematical model and the experimental data, the pulsatile change of the radius does not affect the blood flow velocity.

As for the dependency of the radius on the distance from the entry zone a quantitative analysis had not done until only recently. Melbin (1981) [27] firstly simulated the radius of the femoral artery by nonlinear model. For the compartment of distance 10cm, the internal radius could be expressed as $r(x) = 0.23 * cm * \exp(-0.02\ x)$. Thererfore the exponential approximation could be applicable. On the other hand in their linearlyzed model r equals 0.22cm. Even thought the given distance was short, the tapering constant -0.02 makes the these difference negligible.

Attinger (1967) [28] also approximated the biophysical structural properties of the canine arterial system by the exponential functions. For the femoral artery the resistance was $R(x)=174*\exp(0.098x)$ dyn/cmsec, the leactance was $L(x)=35.5\exp(0.052x)$ dyn/cmsec, the compliance was $C(x)=0.78\exp(-0.076x)$ dyn/cmsec. These approximation were remarkably high quality. However these complicated exponential and non linear approach gives no advantage for constructing simple comprehensive mathematical model. Nevertheless the exponential distributed model affords us much informations for the case of the modeling of the aortic arch. In such a situation simple linear equations does not hold. A further complicated Ricatti type nonlinear differential equation must be used. We show this type of model in the following papers.

VII. The separation of the flow

The separation of the flow occurs in the region such as the post stenotic dilatation where the abrupt change of the pressure gradient exist. In the tube having a nonstenotic constant radius, the pressure gradient dP/dx is negative. However in the down stream of the post stenotic region, the stream line diverge. In such a situation the pressure gradient dP/dx become positive. Then the state is called in the adverse pressure gradient. Therefore the velocity of the particle in the fluid decrease especially at the neighbour of the arterial wall where the viscous retardation force decelerate the fluid movement and make the direction inversed. This inversed flow conflict with the following forward stream. Then the separation of the flow occurs. Increasing the Reynolds number causes the enlargement of the area of the separation where the vortex or even turbulence developes. However in the actual normal artery which have only slight tapering, The stenosis is negligible. In addition the critical Reynolds number in the pulsatile flow is extremely larger than that of the steady flow. Therefore the separation of the flow can be neglected.

VIII. The secondry flow

In many shapes of the cross section except circule or in the bending tube, the flow never attains axisymmetric flow and bears cubic deformative changes. In such a case the inertia acts as a centrifugal force from the central part of the flow in the tube to the lateral part of the tube centrifugally. As the velocity profile of the artery for the radial direction is conical, the velocity should attain the maximum value at the axis of the flow and minimum at the wall. Therefore larger centrifugal force exist near the axis rather than in the neighbour of the wall. The pressure gradient operate so that balance out such unevenly distributed centrifugal forces. Near the arterial wall where the flow velocity is small, the pressure gradient is larger than the centrifugal force. Consequently there developes another flow which direct toward the central axis of the tube. This is the secondary flow. Even in the straight tube, if the shape of the cross section is not circule, an ene-

vitable seconary flow happens. In the human arterial system, strictly speaking the cross section is never circle and certain secondary flow may exists. However in some compartment such as femoral artery, the change of the cross section area is small and the flow in such a space can be regarded to have only axial component. In our model therefore the secondary flow was neglected.

IX. The effect of the entry zone

The flow velocity profile differences exist between the entry zone (inlet zone) and the more down stream region. Especially for the viscous fluid, as the flow moves toward the down stream the thickness of the boundary layer increase. Then the effect of the fluid viscousity developes for the whole plane of the tube. The compartment to which the effect of the fluid viscousity reach for the whole cross section is called the entry zone. After this compartment the velocity profile become stable. In the case of the laminar flow, the inlet length of the entry zone is approximated as $0.065 * (Re) * S$. For example in the femoral artery of the steady flow, assuming that $Re = 500$, $D = 0.5$cm, then the length is 48.75cm. So the effect of the entry zone covers almost whole length of the arterial segment. Such a calculation holds only for the steady laminar flow in the rigid tube. The equation would be far more complex for the case of pulsatile flow in the viscoelastic tube where the flow pattern is not laminar. Therefore we do not consider the effect of the entry zone.

X. The geometric character of the arterial system

The geometric branching of the arterial system are known to obey the experience law of Roux.

1) The symmetric branching

2) The branching angle of the small artery is larger than that of the large artery.

3) The total cross sectional area of the after branching artery are larger than that of the stem artery.

4) In the symmetric branching, the diameter of the branched artery is smaller in 20-30% than the diameter of the stem artery.

In the actual arterial system, there have many branching points. So the pulse wave bears reflection from many points along the artery. To include such terms makes the model comple and the mathematical treatment is inoperable. We only adopped the case of straight axisym ic circular tube and the reflection point are confined to the terminal point only. The reflection of the pulse wave is refered in the following paper.

XI. The non linear term of the Navier-Stokes equation

The convective acceleration terms are inherent physical quantity which operate in the moving fluid. This acceleration do exists even the flow is stational. Since the term Dv/Dt express the change of the velocity in the time domain at the local flow field. The existence of this term indicate

the unsteady flow. On the other hand the nonlinear term in the Lagurange's differentiation (the material derivation) singifies the convective change of the flow. This is the intrinsic acceleration which developes when the fluid transfer in the space domain where the physical quantity such as the flow velocity distribute unevenly. This non linear terms are the essential difference from the dynamic of the rigid material. The nonlinearity of the Navier-Stokes equation originates in this convective acceleration terms. Womerseley (1958) examined the effects of these nonlinear terms on the mean longitudinal flow velocity with the help of the first order perturbation correction method. He incorporated only two nonlinear term $u * \alpha u/\alpha r$ and $w * \alpha w/\alpha z$. To clarify the effects of the nonlinear terms he used the Stokes's stream function. The form of the additional term which were induced by the nonlinear terms included the higher order products of the Bessel functions. Therefore the finite integration of these terms became negligible small. The corrected axial flow velocity increased only 5% (7cm/sec). This value is over estimated for he assumed Co (the pulse wave velocity) $=500$cm/sec. Melbin (1981) calculated the contribution of each terms of the Navier-Stokes equation to the blood pressure in the human femoral artery. The terms $w * \alpha w/\alpha z$, $u * \alpha w/\alpha r$ contributed only 5.6%, 2.4% respectively. The proportion was extremely small in comparison with the main linear term. The convective accelerative terms as has been mentioned express the essential and intrinsic characteristic properties which only the fluid itself have. However in the practical blood flow, contribution of these terms seemed to be small 5% at best. We have neglected these nonlinear terms because of such tentative minor contributions. Nevertheless it is suspicious to remove the nonlinear terms for the sake of the appropriate expression of the blood flow. The interaction between these nonlinear terms or between the linear terms may produce an unexpected unknown effect on the flow. The numerical solution of the Navier-Stokes equation have recently been reported in the field of the engineering by utilyzing the computer technique. However these methodlogical problems are out of our duty.

XII. The significance of the Stokes stream function

The stream function ψ is the covariant function with the velocity potential function ϕ. The stream function maintains a constant value along the line of the stream whose direction coinsides with the direction of the tangent at the arbitrary point on the given curves. In another words, the line on which the stream function keeps the constant value represent the line of the strea in the plane. In the incompressible plane fluid without vortex, the rotation of the velocity vector is zero. Then $\alpha v/\alpha x - \alpha u/\alpha y = 0$. Utilizing the velocity potential $u = \alpha \phi/\alpha x$, $v = \alpha \phi/\alpha y$, the velocity components are expressed by the differentiation of the velocity potential. Further more the fluid is incompressive where there is no divergence, then the $\mathrm{div}(v) = 0$. So

$\alpha u/\alpha x + \alpha v/\alpha y = 0.$

Now setting the covariant function with the velocity potential as

$\alpha \phi/\alpha x = \alpha \psi/\alpha y, \qquad \alpha \phi/\alpha y = -\alpha \psi/\alpha x$

Then the each velocity compnents are represented by these stream function as

$u = \alpha \psi/\alpha y \qquad v = -\alpha \psi/\alpha x$

So to utilyze the steam function one should permit the following condition of the flow

1. No vortex (the rotation of thr vector is zero)
2. The fluid is incompressible
3. No divergent flow

In our theoretical expansion we have already made such assumptions. Therefore the stream function should be applicable for our study.

In this paper we have obtained the pure blood flow velocities from linearlyzed Navier-Stokes equations. To construct the distributed parameter model, one should incorporate into the transmission line equation into not only the blood properties but also the mechanical properties of the arterial wall. In the following two papers, we shall expand the constructive dynamical analysis of the arterial wall.

Referances

1. Womersley JR (1958) An elastic theory of pulse transmission.WADC.TR 56-614.
2. Westerhoff N, Bosman F, DeVries CJ, Noordergraaf A (1968) Analog study of the human systemic arterial tree. J.Biomechanics 2:121-143.
3. Womersley JR (1954) Oscillatory motion of a viscous liquid in a thin walled elastic tube. Phil.mag.Ser 46:199-220.
4. Jager GN, Westerhoff N, Noodergraaf A (1965) Oscillatory flow inpedance in electrical analog of arterial system. Cir.Res 16:121-140.
5. Westerhoff N, noodergraaf A (1970) Arterial viscoelasticity. A generalyzed model. J.Biomechanics 3:357-379.
6. Karreman G (1952) Some contributions to the mathematical biology of blood circulation. Reflection of pressure wave in the arterial system. Bull.Math.Biophys 14:327-350.
7. Benis AM, Usami S,Chien MB (1970) Effect of hemnatocrit and inertial loss on the pressure-flow relations in the isolated hind paw dog. Cir.Res 27:1047-1068.
8. Brooks DE, Goodwin JW, Seamam GVF (1970) Interaction among etythrocites under shear. J.Apply.Rhysiol 28:172-177.
9. Whitmore RL (1968) Rheology of the circulation. Pregamon Press, Oxford.
10. Bayliss LE (1965) Handbook of physiology. circulation I.chap 8.
11. Haynes RH, Burton AC (1959) Role of the non-newtonian behaviour of blood in hemodynamics. AM.J.Physiol 197:943-950.

12. Sarpkaya T (1966) Experimental determination of critical Reynolds number for pulsatile poiseuille flow, trans. ASME.J.Basic.ENG 88:589-598.
13. Hino M, Sawamoto M, Takasu S (1976) Eperiments on transmission to turbulence in an oscillatory pipe flow. J.Fluid.Mech 75:193-207.
14. Yellin EL (1966) Laminar turbulence transition process in pulsatile flow. Cir.Res 19:791-804.
15. Hale JF, McDonald DA, Womersley JR (1955) Velocity profiles of oscillating arterial flow with some calculation of viscous drags and Reynolds number. J.Physiol 128:629-640.
16. McDonald DA (1952) The velocity of blood flow in the rabbit aorta studied with high speed cinematography. J.Physiol 118:328-339.
17. Dick DE, Kendrick JE, Matson GL (1968) Measurement of nonlinearity in the arterial ayatem of the dog by a new method. Cir.Res 22:101-111.
18. Barnett GO, Mallos AJ, Shapiro A (1961) Relationship of aortc pressure and diameter in dog. J.Apply.Physiol 16:545-548.
19. Patel DJ, DeFreitas FM, Fry DL (1963) Relation of radius to pressure along the aorta living dog. J.Apply.Physiol 18:1111-1120.
20. Patel DJ, Austen WG, Greenfield J (1964) Imoedance of certain large veddels inman. ANN.N.Y.Aced.Sci 115:1129-1139.
21. Arndtt JO, Klauske J, Mersh F (1968) The diameter of the intact carotid artery in man and its change with pulse pressure. Pfluger.Arch 301:230-240.
22. Cox RH (1975) Pressure dependance of the machanical property of arterial wall. Am.J.Physiol 229:1371-1375.
23. Tickner EG, Sacks AH (1967) A theory for the static elastic behaviour of blood veseel. Biorheology 4:151-168.
24. Cox RH (1975) Arterial wall mechanics compositionand effects of smooth muscle activation. Am.J.Physiol 229:807-812.
25. Cox RH (1976) Mechanics of canine illiac arterial smooth muscle in vivo. Am.J.Physiol 230:462-470.
26. Attinger EO, Anne A, Sugawara H, Mikami T (1966) Analysis of pulsatile blood flow through a system of branched non uniform tube. Abstract. ANN.Symp.Biomath.Comp.Sci.4th. Houston. Texas.
27. Melbin JK, Riffle RA, Noodergraaf A (1981) Pulse wave transmission. Cir.Res 49:442-452.
28. Attinger EO, Sugawara H, Navarro A, Ricceto A, Martin R (1966) Pressure flow relation in dog arteries. Cir.Res 19:230-246.