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Finite Difference Method for Mechanical Adaptation of Arteries to Sustained Hypertension

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ABSTRACT

The dynamics of arterial wall remodelling under hypertensive conditions is discussed here. Sustained hypertension was simulated by a step increase in blood pressure. The arterial wall was considered to be a thick walled tube made of non linear elastic incompressible material the driving stimuli for the geometric adaptation are the normalized deviations of wall stresses from their values under normotensive conditions. Meachanical adaption is driven by the difference between the area compliance under hypertensive and normotensive conditions. The predicted time course of the geometry and mechanical properties of arterial wall are in good qualititative agreement with published findings. Crank Nicolson finite difference scheme is used for computation purpose which is fast conversing in comparision to the method used by Rachev et al (1998).

Keywords- Hypertension, Adaptation, Incompressible.

I. INTRODUCTION

There exists an increasing interest in the investigation of changes that appear in the geometry structure and mechanical properties of the arterial wall when the vessels are subjected to abnormal pressure and flow conditions (Rachev et al 1998). Understanding of the processes of arterial adaptation is of importance because it has been postulated that some pathological states might be considered as a result of remodelling gone into wrong direction (fung et al. 1993)

Experimental studies on geometric and oral recognition of terraneral parels Ave been carried out it several directions bors have tidied De adaptive response of the vascular wall to change in bed flow increase of the wall thickness and a reduction of the il riu lengphudimal thretch ratio of dogs aasta due to sustained hypenension has been observed by vaishnave (1990)

Coupling benveen grametric and mechanical moshuarities in the process of arterial wall deformation enhances the complexity in the adaptation proosdire described above. Thickening of the arterial wall in response to increased pressure causes a change of the defensed arterial radius. Then, even when the flow is kept constant, the shear stress at the arterial endothelium is altered which engages mechanisme for the adaptation of the inner radius. On the other hand any change of mechanical properties of the vascular material causes changes in the stress and strain distribution in the arterial wall even when the artery is subjected to constant fond.

An efficient approach to better understanding and assessment of the contribution of different factors involved in arterial wall as well as interrelation between them, in the mathematical modelling and numerical simulation of the process of remodelling under altered pressure or flow conditions

A different approach to study the dynamics of the geometric remodelling in response to changes in blood pressure has been applied by Rachev et al (1996).

They studied the dynamics of geometric adaptation in response to change in blood pressure. It was assumed that during remodelling the zero-stress configuration of the arterial cross section remains a circular sector. The wall changes only its geometric

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dimension while the material properties remain the same.

Governing Equations:

For incompressible wall material and radial, circumferential and axial stretch ratio are given by

$$R_i = \frac{H}{m-1},\ R_0 = \frac{mH}{m-1},\ \phi = \pi - \frac{L_0 - L_i}{2H} \quad \text{where } m = \frac{L_0}{L_i} \qquad \text{.....(1)}$$

$$\lambda r = \frac{R}{\lambda_z r} \qquad \lambda_\theta = \frac{\chi_i}{R} \qquad \lambda_z = \lambda \qquad \text{.....(2)}$$

where $\chi = \pi/\pi - \phi$, r and R are the radial co-ordinates of an arbitrary point in the deformed and zero stress state respectively, being related through the equation

$$\gamma = \sqrt{\frac{R^2}{\lambda_z} + R_i^2\left(\mu^2 - \frac{1}{\lambda_z}\right)} \qquad \text{.....(3)}$$

The deformation parameters μ and λ are defined as

$$\mu = \frac{r_i}{R_i} \qquad \lambda = \frac{l_z}{L_z} \qquad \text{.....(4)}$$

with r_i being inner radius in the deformed state and I_L and L_t being the length of the arterial segment at deformed and zero-stress state respectively The non vanishing components of the green strain tensor are

$$e_j = \frac{1}{2}(\lambda_j^2 - 1)(j = r, \theta, z)$$

The radial , circum ferential and longitudinal stresses are given by

$$\sigma_r = \lambda_r^2 \frac{\partial w}{\partial e_i} + q \qquad \sigma_\phi = \lambda_\phi^2 \frac{\partial w}{\partial e_\phi} + q \qquad \sigma_z = \lambda_z^2 \frac{\partial w}{\partial e_z} + q \qquad \text{.....(6)}$$

where W(e_r, e_O, e_z) is the strain energy function which completely describe the mechanical properties of the wall material q is the unknown scalar function that has to be determined from the equilibrium equation and boundary conditions.

After integration of the differential equations of equilibrium and imposing the boundary conditions for the internal surface sigma $_R$(r = r_1) = - p and the outer surface sigma $_r$ (r = r_O) = 0 the following expression for the wall stresses are obtained

$$\sigma_r = \int_{ri}^{r} \left(\lambda_\phi^2 \frac{\partial \omega}{\partial e\phi} - \lambda_r^2 \frac{\partial \omega}{\partial e_i}\right)\frac{dr}{r} - p$$

$$\sigma_\theta = \sigma_r + \lambda_\phi^2 \frac{\partial \omega}{\partial e_\phi} - \lambda_r^2 \frac{\partial \omega}{\partial e_r} \qquad \text{.....(7)}$$

$$\sigma_z = \sigma_r + \lambda_z^2 \frac{\partial \omega}{\partial e_z} - \lambda_r^2 \frac{\partial \omega}{\partial e_i}$$

where r_i and $r_O$are the deforined inner and outer radius, respectively Pressure p and the axial force $F_z$relate to wall stresses given below.

$$p = \int_{ri}^{ro} \left(\lambda_\theta^2 \frac{\partial \omega}{\partial e_\theta} - \lambda_r^2 \frac{\partial \omega}{\partial e_r}\right)\frac{dr}{r} \qquad F_z = 2\pi \int_{ri}^{ro} r\sigma_z dr \qquad \text{.....(8)}$$

The Everage Cicrcum ferential axial sresses are given below

$$\sigma_{\theta av} = \frac{p_{ri}}{r_0 - r_i} \qquad \sigma_{zav} = \frac{F_z}{\pi\left(r_0^2 - r_i^2\right)} \qquad \text{.....(9)}$$

The mean shear stress at the inner arterial surface is given by poiseuille,s law.

$$\tau = \frac{4\eta Q}{\pi r_i^3} \qquad \text{.....(10)}$$

where Q is flow and η blood viscosity.

II. REMODELLING RATE EQUATIONS

Following Rachev et al (1998) the artery is first considered under normotensive conditions. The dimensions at the zero stress state are LN. L. L and HN. Superscripts N and H denote the values under normotensive and hypertensive conditions respectively, the vessel is subjected to normal blood pressure p" and is kept at constant deformed length Induced arterial hypertension is modeled by a step increase in blood pressure from pt to ph. Blood flow rate is kept constant.

To monitor the remodelling of zero-stress state, the following growth parameters are defined.

$$\alpha(t) = \frac{L_i^H(t)}{L_i^N} \quad , \quad \beta(t) = \frac{L_0^H(t)}{L_0^N}$$

$$\gamma(t) = \frac{H^H(t)}{H^N} \quad , \quad \delta(t) = \frac{L_z^H(t)}{L_z^N} \qquad \text{.....(11)}$$

It is postulated that the artery remodels its zero-stress configuration in a manner to restore the distribution of circumferential stress and the magnitude of the average axial stress as they are under normotensive condition. Since circumferential stresses vary smoothly through out the arterial wall the following remodeling rate equation for the growth parameters are postulated.

$$\frac{d\alpha}{dt} = \frac{1}{t_L}\left[\frac{\sigma_i^H(t) - \sigma_i^N}{\sigma_i^N}\right] \qquad \text{.....(12)}$$

$$\frac{d\beta}{dt} = \frac{1}{t_L}\left[\frac{\sigma_0^H(t) - \sigma_0^N}{\sigma_0^N}\right] \qquad \text{.....(13)}$$

$$\frac{d\gamma}{dt} = \frac{1}{t_H}\left(\frac{\sigma_{\theta av}^H(t) - \sigma_{\theta av}^N}{\sigma_{\theta av}^N}\right) \qquad \text{.....(14)}$$

$$\frac{d\delta}{dt} = \frac{1}{t_z}\left(\frac{\sigma_{zav}^H(t) - \sigma_{zav}^N}{\sigma_{zav}^N}\right) \qquad \text{.....(15)}$$

$$\frac{\partial \sigma_{\theta av}^H}{\partial c}\frac{dc}{dt} + \frac{\partial \sigma_{\phi av}}{\partial b_i}\cdot\frac{db_1}{dt} + \frac{\partial \sigma_{\theta av}^H}{\partial b_1}\cdot\frac{db_1}{dt} = 0 \qquad \text{.....16}$$

$$\frac{\partial \sigma_{zav}^H}{\partial c}\frac{dc}{dt} + \frac{\partial \sigma_{zav}^H}{\partial b_1}\cdot\frac{db_1}{dt} + \frac{\partial \sigma_{zav}^H}{\partial b_1}\cdot\frac{db_1}{dt} = 0 \qquad \text{.....17}$$

where $\sigma_i^H(t)$, $\sigma_0^H(t)$ $\sigma_\theta^H av(t)$ and $\sigma_z^H av(t)$ are the current cicum-ferential stress at the inner surface. The stress at the outer surface, the average circum ferential and the average axial stress of the hypertensive artery σ_i^N. σ_0^N $\sigma_\theta^N av$ and $\sigma_z^N av$ are corresponding stress in the normotensive artery t_L , t_H and t_z are characterstic time constants

c_A^N and $c_A^H(t)$ are the area compliance

$c_A = \frac{\Delta A}{A \Delta P}$. (Cow and Taylor 1960)

The mismatch between these compliance plays the role of a driving stimulus for the change of the material constant c. Following the introduced hypothesis for the compatibility between geometric and mechanical adaptation. It is accepted that the constant b_1 and b_2 vary in a manner to maintain the current level of the averge circumferential and average axial stress. This implies that at any moment the rate of change of the average stresses with respect to the variables material constant c, b_1 and b_2 respectively which yields

Applying Crank-Nicolson scheme in the above equations, the above equations become in the discretized form as follows:

The aim of this study is to propose a relatively simple but general mathematical model, using Crank Nicolson Scheme which accounts for both the geometric and mechanical remodelling of arteries in response to induced hypertension. Theoretical predictions of the model are compared against available experimental data to assess the validity of certain new hypothesis concerning the driving stimuli and interelation between wall-stress-dependent compliance adaptation.

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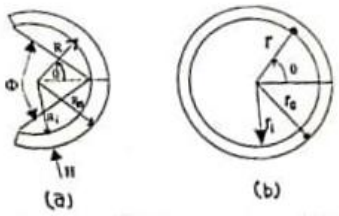

Figure 1. Schematic diagram of an artery at (a) zero-stress state and (b) loaded state

The artery is consider to be a thick walled tube made of non-linear, elastic orthotropic and incompressible material. At the zero stress state the vessel cross -section is considered to be a circular sector with the following dimensions. Inner arc length L_i, outer arc length Lo and thickness H. the inner and outer radii of curvature (R, R) and the opening angle o. are calculated using the formulae.

Schematic diagram of an artery at (a) zero stress state (b) loaded state under physiological loading, arteries are in a finite axisymetric plane strain state. The deformed arterial cross section is shown in fig (b)

$$\frac{1}{4\Delta c}\left(\overset{H}{\sigma}{}_{\theta}{}^{k+1}_{i} - \overset{H}{\sigma}{}_{\theta}{}^{k}_{i}\right)\frac{1}{\Delta t}\left(c^{k+1}_{i+1} - c^{k+1}_{i-1} + c^{k}_{i+1} - c^{k}_{i-1}\right) +$$

$$\frac{1}{4\Delta b_1 \Delta t}\left(b^{k+1}_{1i+1} - b^{k+1}_{1i-1} + b^{k}_{1i+1} - b^{k}_{1i-1}\right)\left[\sigma\phi^{k+1}_{iav} - \sigma\phi^{k}_{i} + \sigma^{H\,k+1}_{\theta iav} - \sigma^{H}_{\theta iav}\right] \quad \text{.....18}$$

$$\frac{1}{4\Delta c}\left(\overset{H}{\sigma}{}_{z}{}^{k+1}_{i} - \overset{H}{\sigma}{}_{z}{}^{k}_{i}\right)\frac{1}{\Delta t}\left(c^{k+1}_{i+1} - c^{k+1}_{i-1} + c^{k}_{i+1} - c^{k}_{i-1}\right) +$$

$$\frac{1}{4\Delta b_1 \Delta t}\left(b^{k+1}_{1i+1} - b^{k+1}_{1i-1} + b^{k}_{1i+1} - b^{k}_{1i-1}\right)\left[\sigma^{k+1}_{ziav} - \sigma z^{k}_{i} + \sigma^{H\,k+1}_{ziav} - \sigma^{H}_{ziav}\right] \quad \text{.....19}$$

III. NUMERICAL METHOD

The system of equations (13) to (19) are highly non linear because all variables depend on complex manner on the geometric parameter of the current zero-stress configuration and elastic properties More over equations (13) to (17) are coupled and have to be solved simultaneously with the equations (2) to (10) for inflation at pressure P ^ H and longitudinal extension 2, at t = 0 when the pressure is increased in step wise manner from P ^ N to P ^ H

The initial value for the growth parameters is alpha = beta = gamma = delta = xi = 1 The values of the inner deformed radius stresses and the compliance C_{A} Change in a stepwise manner when the pressure increase from P ^ N to P ^ H and are calculated using the dimensions of the normotensive artery subjected to the hypertensive pressure at the normotensive in situ, longitudinal axial stretch ratio λ.

The problem of determining the functions a (t) beta(t) * gamma(t) and 8 (t) from the governing equations and initial conditions kappa(0) = beta(0) = gamma(0) = delta(0) = hi a single valued solution making use of equation (1) it is possible to determine that time variations of the dimensions of the zero stress configuration of the arterial cross section and the longitudinal stretch ratio. Solving equations (18) and (19) it is possible to obtain the time course of the elastic properties of the artery also. The integration of (16) to (19) accounts for the flow-induced adaptation of the artery. Equation (16) & (17) are solved by Crank-Nicolson Scheme.

IV. RESULTS AND DISCUSSION

A numerical study using data available in the literature was performed. Data for zero-stress configuration strain energy function for a rabbit thoracic aorta were taken from the paper chuong and Fung (1986). The dimensions of the zero stress state are Lo = 11.25 mm Li = 9.75mm -1086 degree they were accepted to correspond to the homeostatic case in which the artery is subjected to an arterial pressure of P^ prime prime = 13.33k*P_{4} and in situ axial stretch of the material constants c = 22.4 kpa b_{1} = 1.0672 , b_{2} = 0.4775 , b_{3} = 0.0499 b_{4} = 0.903 b_{z} = 585 dot y - x =1.6 b_{s} = 0.0042 for purpose qualititaive analysis. (See Rachev et al (1998)), induced hypertension is simulated by a step increase to a pressure of p ^ 11 = 21.33 kpa.

The present study predicts a monotonic steady state approximately 50% higher than the thickness of the normotensive aorta. The time course of the opening angle shows very rapid increase, reaching a maximum, after which the angle decrease to an asymptotic value.

The present study utilizes a phenomenological model for wall remodelling in which stress-driven processes at the molecular cellular and tissue levels are all lumped in to remodelling rate equations linking the cause (stresses) and the effect (remodelling) this is of course, an over simplifications of the underlying biology in order to yield mathematically simple models. In reality, however the adaptation to stress and wall shear are fundamentaly different, wall stress induces growth principally through smooth muscle hypertrophy, while increased initial shear leads to proliferation of smooth muscle cells.

The pressure-outer radius relationships of Fig. 2(a) show the theoretical prediction of the response of the normotensive and adapted hypertensive aorta inflated at in situ length. The curves represent the mechanical behaviour of the vessels incorporating both the inherent elastic properties of the vascular material and the geometric dimensions at the zero-stress state. The results are in good agreement with experimental findings of Matsumoto and Hayashi (1994) and Rachev at. el (1998).

The use of Crank Nicolson Scheme saves a lot of computer time. These simulation have been performed in Pantium II. Linearized mechanical response of the arteries expressed in terms of the

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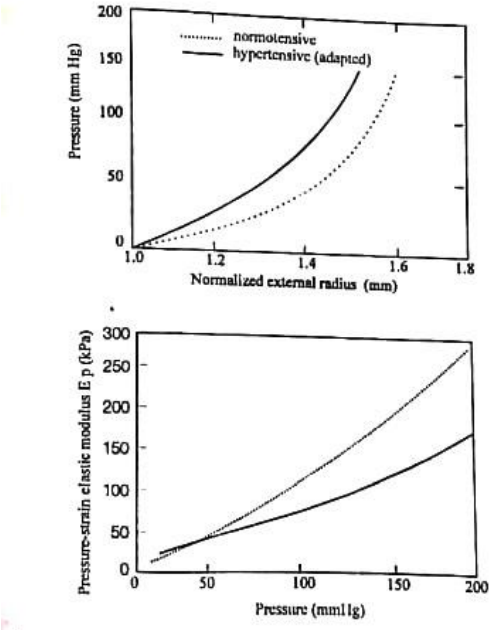
Peterson pressure-strain elastic modulus is shown in fig. 2(b).



Figures 2: (a) and 2(b) illustrate the time variations of the comploiance, C_{A}, and Peterson modulus, E_{p} respectively

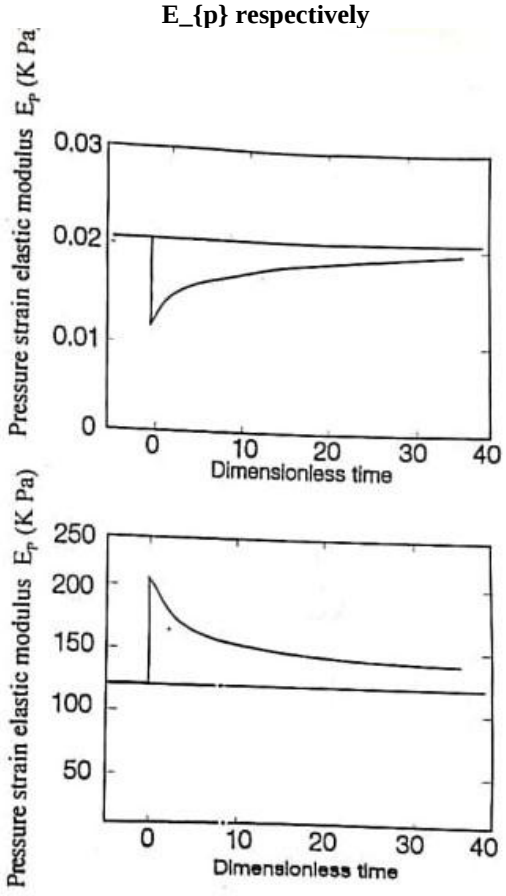


Fig. 3 (a) Pressure-normalized outer radius curves of the normotensive and hypertensive (adapted) norta; (b) Pressure-strain elastic modulus versus pressure for normotensive and hypertensive (adapted) aorta

In brief the proposed scheme is found simple and most satisfying.

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