REVISION
Residual Stresses in Oscillating Thoracic Arteries Reduce Circumferential Stresses and Stress Gradients

H. R. Chaudhry
Department of Mathematics
Center for Applied Mathematics and Statistics
New Jersey Institute of Technology
Newark, NJ 07102

Department of Physical Medicine and Rehabilitation
UMDNJ-New Jersey Medical School
Newark, NJ 07102

B. Bukiet
Department of Mathematics
Center for Applied Mathematics and Statistics
New Jersey Institute of Technology
Newark, NJ 07102

A. Davis
Department of Physical Medicine and Rehabilitation
UMDNJ-New Jersey Medical School
Newark, NJ 07102

A. B. Ritter
Department of Physiology
UMDNJ – New Jersey Medical School
Newark, New Jersey 07103

T. Findley
Neuromuscular Institute
UMDNJ – School of Osteopathic Medicine
Stratford, New Jersey 08084 and
Center for Applied Mathematics and Statistics
New Jersey Institute of Technology
Newark, NJ 07102

(send all correspondence to B. Bukiet, Tel: (201)-596-8392, Fax: (201)-596-6467 e-mail: bukiet@shock.njit.edu)
Keywords: Residual Stresses, Oscillations, Arteries, Atherosclerosis

REVISION

Residual Stresses in Oscillating Thoracic Arteries Reduce Circumferential Stresses and Stress Gradients

H. R. Chaudhry [1, 2] B. Bukiet [2] A. Davis [1] A. B. Ritter [3,] T. Findley [2, 4]

(send all correspondence to B. Bukiet, Tel: (201)-596-8392, Fax: (201)-596-6467, e-mail: bukiet@shock.njit.edu)

Abstract

The purpose of this paper is to examine the effects of residual stresses and strains in the oscillating arteries on the stress distribution in the vascular wall. We employ a static theory of large elastic deformations for orthotropic material (Chuong and Fung, 1986) with the acceleration term added to make the theory dynamic. We use the static elastic parameters of residual stresses in our analysis because the dynamic parameters are not available in the literature. Our analysis reveals that the effect of considering the residual stresses is to decrease the very large circumferential stresses at the inner wall by 62% and reduces the stress gradient through the arterial wall by 94% compared to the case when residual stresses are ignored. Thus, because the arteries do contain residual stresses, the consequent lower stresses at the inner wall and the reduced stress gradient, may reduce the progression of atheroma. Our computations show that the stress gradients do not depend on the heart rate.

[1]Department of Physical Medicine and Rehabilitation, UMDNJ-New Jersey Medical School, Newark, NJ 07102

[2]Department of Mathematics Center for Applied Mathematics and Statistics, New Jersey Institute of Technology, Newark, NJ 07102

[3]Department of Physiology, UMDNJ-New Jersey Medical School, Newark, NJ 07102

[4]Neuromuscular Institute UMDNJ-School of Osteopathic Medicine Stratford, NJ 08084

Introduction

It has been well documented that morphological changes such as dilation of large arteries and thickening of the arterial wall as well as increased arterial stiffness are the results of fatigue of the load bearing elastin fibers due to cyclic oscillating stresses. The replacement of elastin with collagen during remodeling and calcification of elastin and collagenous material (Nichols and O'Rourke, 1990, p. 399) also contribute to increased stiffness. Recent measurements suggest that plaques tend to form in areas where mean stresses at the luminal surface of the wall are low and where the mean stress oscillates around zero one or more times during the cardiac cycle (Fry, 1969; Asakura and Karino, 1990; Moore *et al.*, 1992; Okano and Yoshida, 1992). On the other hand, low stress levels in the arterial wall slow the progression of atherosclerotic plaques (Nollert *et al.,* 1992). Information about the distribution of stresses in the arterial wall is also important in the design of artificial grafts (Schwartz *et al.*, 1992). Direct measurement of arterial wall stress is difficult, invasive or not available; therefore mathematical analysis of stresses and strains is useful in predicting stress and strain distribution throughout the arterial wall.

In previous studies where oscillating stresses in arteries are analyzed (Demiray and Vito, 1983; Misra and Chakravarty, 1982; Patel and Fry, 1966; Singh and Devi, 1990; Vorp *et al.* 1995), the presence of residual stresses are ignored. Choung and Fung (1986) have demonstrated the existence of residual stresses in unloaded intact arteries. That is, when an unloaded intact artery is cut longitudinally, the vessel wall springs open with its cross section becoming a sector (see Fig. 1) . We, therefore, in this paper investigate whether residual stresses of the magnitude occuring in arteries substantially reduce or increase wall stresses. This is important since stress reduction is one of the major factors in retarding atherogenesis and the blood pressure rise in hypertension.

Method

We assume the artery to behave as an orthotropic, homogeneous, incompressible and highly elastic material and use a static theory of large elastic deformations for orthotropic materials (Chuong and Fung, 1986) with the acceleration term added to make the theory dynamic. The artery is further assumed to be a uniform and thick circular cylindrical shell. Although tapering occurs in blood vessels, this analysis can be localized to a small segment in which the taper is negligible (Sobin and Tremer, 1980). It is also assumed that the artery remains symmetric under physiological loading which consists of an inflating pressure and extending force. Thus, its motion may be described using a cylindrical coordinate system by

$$r = r(R,t); \quad \theta = \alpha\Theta; \quad z = \lambda_z(t)Z \tag{1}$$

where (R, Θ, Z) and (r, θ, z) are coordinates in the undeformed reference state 0 and deformed states (1 and 2) respectively, $\alpha = \pi/\Theta_0$ where Θ_0 is the angle shown in the stress free reference state 0 in Fig. 1 and $\lambda_z(t)$ is the dynamic extension ratio in the axial direction. Since dynamic values of λ_z are not available in the literature, we assume λ_z to be constant as in (Demiray and Vito, 1983; Singh and Devi, 1990). The principal stretch ratios in the radial, circumferential and axial direction are, respectively

$$\lambda_r = \frac{\partial r}{\partial R}; \quad \lambda_\theta = \alpha r/R; \quad \lambda_z = \frac{\partial z}{\partial Z} \tag{2}$$

The incompressibility condition $\lambda_r \lambda_\theta \lambda_z = 1$ requires that the relation between r and R has the following form

$$r = \left[\frac{R^2}{\alpha \lambda_z} + A(t) \right]^{1/2} \tag{3}$$

where $A(t)$ is an unknown function which must be determined to evaluate the deformation $(r - R)$ from (3). In this connection, the rhythmic motion of the artery is approximated by the function (Demiray and Vito, 1983)

$$A(t) = A_0 + A_1 sin^2 \omega t \tag{4}$$

where A_0 and A_1 are constants which must be determined and ω is the angular frequency of the heartbeat. Since $\sin^2 \omega t$ is a positive quantity, A_0 is associated with the deformation at the lowest pressure level, i.e. $t = 0$, diastolic, and $A_0 + A_1$ is associated with the deformation at the highest pressure level, i.e. $\omega t = \pi/2$, systolic. Thus, if a and b are the values of r at the inner surface of the artery in diastole and systole, respectively, then using (3) we have (by setting $r_i = a$ for $t = 0$ and $r_i = b$ for $t = \frac{\pi}{2\omega}$)

$$A_0 = a^2 - R_i^2/(\alpha \lambda_z); \quad A_1 = b^2 - a^2 \tag{5}$$

Here the subscript i denotes the inner surface of the artery. Thus, using (3) through (5), the relation between the undeformed and deformed radii is given by

$$r = \left[\frac{R^2}{\alpha \lambda_z} + (a^2 - \frac{R_i^2}{\alpha \lambda_z}) + (b^2 - a^2) sin^2 \omega t \right]^{1/2} \tag{6}$$

Since the vascular tissue behaves as a hyperelastic material, it possesses a strain energy function. We use the pseudo-strain energy function, W, developed by Chuong and Fung (1986) for the constitutive relationship for an orthotropic thoracic artery of the rabbit, by taking into account the effects of residual stresses:

$$\rho_0 W = \frac{c}{2} e^Q \tag{7}$$

where

$$Q = b_1 E_\theta^2 + b_2 E_z^2 + b_3 E_r^2 + 2b_4 E_\theta E_z + 2b_5 E_z E_r + 2b_6 E_r E_\theta \tag{8}$$

and $c = 22.40$ kPa, $b_1 = 1.0672$, $b_2 = 0.4775$, $b_3 = 0.0499$, $b_4 = 0.0903$, $b_5 = 0.0585$ and $b_6 = 0.0042$. Here, E_θ, E_z and E_r are Green's strain components in the circumferential, axial and radial directions, respectively. These are related to the principal stretch ratios by

$$E_i = \frac{1}{2}(\lambda_i^2 - 1) \quad (i = r, \theta, z) \tag{9}$$

As the material is assumed to be incompressible, the strain energy function for the incompressible material is taken to be

$$\rho_0 W^* = \rho_0 W + \frac{H}{2}[(1 + 2E_\theta)(1 + 2E_z)(1 + 2E_r) - 1] \tag{10}$$

where $\rho_0 W^*$ is the strain energy function for the incompressible material and H is the Lagrange multiplier. The Cauchy stress components t_{ij} are then given by

$$t_{ij} = \frac{\rho}{\rho_0} \frac{\partial x_i}{\partial X_\alpha} \frac{\partial x_j}{\partial X_\beta} \frac{\partial}{\partial E_{\beta\alpha}} \rho_0 W^* \qquad (i, j, \alpha, \beta = r, \theta, z) \tag{11}$$

where X_α denotes coordinates of the material point in the reference state and x_i denotes the coordinates in the deformed state; ρ and ρ_0 are the densities at the material points in the deformed and undeformed states, respectively. Using (10) and (11) in the above formula, the stress components equations are given by

$$\begin{aligned} t_{rr} &= F_1(E_r, E_\theta, E_z) + H \\ t_{\theta\theta} &= F_2(E_r, E_\theta, E_z) + H \\ t_{zz} &= F_3(E_r, E_\theta, E_z) + H \end{aligned} \tag{12}$$

where H is the hydrostatic pressure due to incompressibility and

$$
\begin{aligned}
F_1 &= c(1 + 2E_r)[b_6 E_\theta + b_5 E_z + b_3 E_r]e^Q \\
F_2 &= c(1 + 2E_\theta)[b_1 E_\theta + b_4 E_z + b_6 E_r]e^Q \\
F_3 &= c(1 + 2E_z)[b_4 E_\theta + b_2 E_z + b_5 E_r]e^Q \qquad (13)
\end{aligned}
$$

The only nonvanishing equation of motion in cylindrical coordinates with zero body forces is given by

$$\frac{\partial}{\partial r} t_{rr} - \frac{1}{r}(t_{\theta\theta} - t_{rr}) = \rho \frac{d^2 r}{dt^2} \qquad (14)$$

Using (6), we get

$$\frac{d^2 r}{dt^2} = \omega^2 (b^2 - a^2) \left[\frac{1}{r} cos2\omega t - \frac{b^2 - a^2}{4r^3} sin^2 2\omega t \right] \qquad (15)$$

Introducing (15) into (14) and integrating, we get

$$
\begin{aligned}
t_{rr} &= \int^r \frac{1}{r} F(E_r, E_\theta, E_z) dr \\
&+ \rho\omega^2 (b^2 - a^2) \left[cos2\omega t \times ln(r) + \frac{b^2 - a^2}{8r^2} sin^2 2\omega t \right] + K(t) \qquad (16)
\end{aligned}
$$

where $F(E_r, E_\theta, E_z) = F_2(E_r, E_\theta, E_z) - F_1(E_r, E_\theta, E_z) = t_{\theta\theta} - t_{rr}$ and $K(t)$ is a function of t which must be determined from the boundary conditions. Assuming that the surrounding tissues exert a pressure $P_0(t)$ on the exterior of the arterial wall, $r = r_o$, we must have

$$t_{rr}|_{r=r_o(t)} = -P_0(t) \qquad (17)$$

Using (17) in (16), we get

$$
\begin{aligned}
-P_0(t) &= \int^{r_0} \frac{1}{r} F(E_r, E_\theta, E_z) dr \\
&+ \rho\omega^2 (b^2 - a^2) \left[cos2\omega t \times ln(r_0) + \frac{b^2 - a^2}{8r_0^2} sin^2 2\omega t \right] + K(t)
\end{aligned}
$$

Therefore,

$$\begin{aligned} K(t) &= -P_0(t) - \int^{r_0} \frac{1}{r} F(E_r, E_\theta, E_z) dr \\ &- \rho\omega^2 (b^2 - a^2) \left[cos2\omega t \times ln(r_0) + \frac{b^2 - a^2}{8r_0^2} sin^2 2\omega t \right] \end{aligned}$$

Substituting this value of $K(t)$ into (16), we get

$$\begin{aligned} t_{rr} &= \int_{r_o}^{r} \frac{1}{r} F(E_r, E_\theta, E_z) dr - P_0(t) \\ &+ \rho\omega^2 (b^2 - a^2) \left[cos2\omega t \times ln(r/r_o) + \frac{b^2 - a^2}{8} sin^2 2\omega t \times (\frac{1}{r^2} - \frac{1}{r_o^2}) \right] \quad (18) \end{aligned}$$

Again assuming that the inner wall, $r = r_i$ is subjected to pressure $P_i(t)$, we obtain from (18), using $t_{rr}|_{r=r_i(t)} = -P_i(t)$:

$$\begin{aligned} P_i(t) &= P_0(t) + \int_{r_i}^{r_o} \frac{1}{r} F(E_r, E_\theta, E_z) dr \\ &+ \rho\omega^2 (b^2 - a^2) \left[cos2\omega t \times ln(r_o/r_i) + \frac{b^2 - a^2}{8} sin^2 2\omega t \times (\frac{1}{r_o^2} - \frac{1}{r_i^2}) \right] \quad (19) \end{aligned}$$

From (12), $t_{\theta\theta} = t_{rr} + (t_{\theta\theta} - t_{rr}) = t_{rr} + F$. Then, using (18), we have

$$\begin{aligned} t_{\theta\theta} &= \int_{r_o}^{r} \frac{1}{r} F(E_r, E_\theta, E_z) dr \\ &+ \rho\omega^2 (b^2 - a^2) \left[cos2\omega t \times ln(r/r_o) + \frac{b^2 - a^2}{8} sin^2 2\omega t \times (\frac{1}{r^2} - \frac{1}{r_o^2} \right] \\ &- P_0(t) + F(E_r, E_\theta, E_z) \quad (20) \end{aligned}$$

and from (12)

$$t_{zz} = t_{rr} + F_3(E_r, E_\theta, E_z) - F_1(E_r, E_\theta, E_z) \quad (21)$$

The external force N at the ends of the arterial tube to maintain this deformation is given by

$$N = 2\pi \int_{r_i}^{r_o} t_{zz} r dr \quad (22)$$

Since the dynamic values of the parameters for the residual stress case are not known, we shall use the results from the static experiment conducted by Chuong and Fung (1986) for the rabbit thoracic artery. The inner radius of the artery in the reference state 0 is $R_i = 3.92$mm. The outer radius of the artery in the reference state 0 is $R_o = 4.52$mm. The inner radius, r_i, in the unloaded state 1 is 1.393 mm, and the outer radius, r_o, is 1.989 mm. The values of a and b, calculated for pressures of 60 mm Hg (diastole) and 120 mm Hg (systole), are 2.400 mm and 2.653 mm, respectively.

In order to calculate a, we use (19) with $\omega = 0$, $P_0(t) = 0$, $P_i(t) = 60$ mm Hg, $r_i = a$ and $r_o = \left[\frac{1}{\alpha\lambda_z}(R_o^2 - R_i^2) + a^2\right]^{1/2}$, (from (6)), for the static case. In this case, since $\omega = 0$, equations (6) and (19) do not contain b.

Similarly, we use $\omega t = \pi/2$, $P_0(t) = 0$, $P_i(t) = 120$ mm Hg, $r_i = b$ and $r_o = \left[\frac{1}{\alpha\lambda_z}(R_o^2 - R_i^2) + b^2 - a^2\right]^{1/2}$, (from (6)), for the static case to calculate b after using the value of a determined above.. The values of a and b are found for the static case so that (19) will be true in both the static and dynamic cases.

We use $\Theta_0 = 71.4^o$, $\alpha = \pi/\Theta_0 = 2.521$ and $\lambda_z = 1.542$ (Chuong and Fung, 1986). From Bulanowski and Yeh (1971) ρ is taken to be 0.9 gm/cm^3.

In the case in which residual stresses are not considered, the following parameters were used for the same artery of the residual stress case (Chuong and Fung, 1983) $R_i = 1.393$ mm, $R_o = 1.989$ mm (in state 1, as states 0 and 1 become identical because there is no reference state 0 in this case. Therefore $R_i = r_i$ (of residual case) $= 1.393$ mm and $R_0 = r_0$ (of residual case) $= 1.989$ mm in the unloaded state), $a = 2.342$ mm, $b = 2.576$ mm (calculated as explained above for the residual stress case) $\Theta_0 = 180^o$, $\alpha = 1$, $c = 26.95$ kPa, $b_1 = 0.9925$, $b_2 = 0.4180$, $b_3 = 0.0089$, $b_4 = 0.0749$,

$b_5 = 0.0295$, $b_6 = 0.0193$ and $\lambda_z = 1.542$.

We have analyzed the results of the existing residual stresses on the dynamic pressure at the inner wall and stresses throughout the wall of the rabbit thoracic artery. Since there is no external pressure on the thoracic artery, as observed physiologically (Guyton and Hall, 1996), we take $P_0 = 0$. Some of the results obtained in this analysis are reproduced below.

Results

The effect of taking into account the residual stresses is to decrease the very large circumferential stress at the inner wall by 62% and reduce the stress gradient at the inner wall by 94% (Fig. 2). It is observed that in the residual case where the ratio $R_i/R_o = .867$, the stress at the inner wall is 240 kPa; the stress gradient at the inner wall is approximately 730 kPa/mm and at the outer wall, it is approximately 286 kPa/mm. Whereas in the absence of residual stresses, where the ratio of $R_i/R_o = .700$, the stress at the inner wall is 636 kPa; the stress gradient at the inner wall is approximately 13000 kPa/mm and at the outer wall, it is approximately 240 kPa/mm. Thus at the outer wall, the stress gradient is almost the same in both the cases. Thus, the existence of residual stresses in arteries serves to lower the stresses and stress gradients at the inner wall, which in turn may reduce the progression of atherosclerotic plaques (Nollert *et al.*, 1992). We have also observed, by varying the heart rate in the computations, that the stress gradients do not vary with changes in heart rate.

Our computations show that the variation of dynamical pressure at the inner wall versus time is the same in the presence or absence of residual stresses (Fig. 3).

We observe that the presence of residual stresses drastically reduces the stresses at the inner wall of the artery. The maximum stress is reduced from approximately 636 kPa to 240 kPa while the minimum stress is reduced from approximately 207 kPa to 86 kPa and these do not depend upon the heart rate (Figs. 4 and 5).

Discussion

Actually, the arterial tissue as observed by Fung, *et al.* (1993), for the case of the rat, remodels itself and its elastic parameters change under stress. However, no systematic measurements have been performed so far on relaxation, creep and hysteresis characteristics of blood vessels during the remodeling process (Fung, *et al.*, 1993) for calculating dynamic elastic parameters. Therefore, in the absence of dynamic elastic parameters, we have used the static parameters in our analysis as given in (Chuong and Fung, 1986). However, the dynamic analysis presented here can be used to find the dynamic elastic parameters by performing the experiment described in (Chuong and Fung, 1986) for static elastic parmeters.

Since the quantitative results are obtained for rabbit thoracic artery, these cannot be generalized to all types of arteries. However, qualitatively the results are valid for any large artery.

We have taken the external pressure for rabbit thoracic artery to be zero. This may not be true for other arteries, such as coronary arteries and arteries embedded in skeletal muscles during moderate exercise. For example, the external pressure for coronary artery is $80-120$ mm Hg and for arteries in skeletal muscle during moderate exercise, it is $10-30$ mm Hg (Guyton and Hall, 1996). This must be taken into account when calculating stresses.

Arterial oscillations are not actually of sinusoidal nature. For mathematical convenience, we have approximated the oscillations by a sinusoidal function. The quantitative results are therefore approximate, although they are qualitatively correct.

By setting $\omega \to 0$ and $\omega t \to \pi/2$ in (19), we get the highest value of the static pressure, P_i^{HS}, i.e., in the systolic state. Again, by setting $t = \pi/(2\omega)$, i.e., one half of the period, we get the highest value of the dynamic pressure, P_i^{HD}, i.e., in the systolic state. Also the relation between P_i^{HS} and P_i^{HD} is given by $P_i^{HD} = -\rho\omega^2(b^2 - a^2)\, ln\, \frac{r_o^H}{r_i^H} + P_i^{HS}$ where r_o^H and r_i^H are the values of radius on the outer and inner surfaces, respectively, which can be obtained from (6). Thus, P_i^{HD} decreases due to the negative inertial force (which is very small compared to P_i^{HS}). This happens immediately after the systolic state starts and the arterial wall begins to experience negative inertial force. This causes backward flow to occur in the early stages of the systolic state for some groups of arteries for a short duration. Therefore, if negative flow occurs in conjunction with negative inertia term, P_i^{HD} decreases but does not become negative, thus preventing arterial collapse in the systolic state.

In the case of arteries, where external pressures exist, the external pressure may be calculated from the formula $P_0(t) = \frac{hYE_\theta(r_o,t)}{r_o(1 - \nu^2)}$ (Misra and Chakravarty, 1982; Misra and Singh, 1984; Rogers and Lee, 1964), where ν, Y and h are Poisson's ratio, the Young's modulus and the thickness of the surrounding connective tissues, respectively. This formula is also helpful in interpreting some results which can be used for the design of artificial arteries and veins. For example, $P_0(t)$ decreases as h/r_0 decreases (other parameters held fixed for a particular species). Therefore, the circumferential stress, as seen from (20) is likely to increase, thus decreasing the resistance to atherogenesis. It is also noted that the value of Θ_0, to be determined experimentally, (see Fig. 1) will vary as Y varies. Consequently , $\alpha = \pi/\Theta_0$ and deformed radius r_0 will change (see (6)). Therefore, h/r_0 should be chosen so that for given values of Y and ν, the calculated value of $P_0(t)$ lies within its physiological range. Therefore, as Θ_0 is not known for other arteries, the effect of varying Y cannot be evaluated in this work. However, if Θ_0 is known, such analysis should be helpful in selecting vascular tissues for autologous grafts. Furthermore, the failure of angioplasty and arterial bypass can be better understood by considering the results of this analysis.

In diseased arteries, such as occur in hypertension and diabetes, the arterial wall thickens, i.e., the ratio of R_i/R_0 decreases. This will result an increase in stresses and stress gradients (see item 1 of the Results section) which will further increase the progression of these diseases. Therefore, this model agrees with the physiological prediction.

We have shown that the presence of residual stresses reduces the computed stresses in arterial walls subjected to oscillating pressures. Residual stresses play a dual role in arterial walls. On the one hand, lower levels of oscillating stress at the luminal walls of arterties help promote atherogenesis since atherosclerotic plaques develop preferentially in the regions of low stress levels that oscillate around zero during the cardiac cycle (Moore *et al.*, 1992). On the other hand, low stress levels in the arterial walls would decrease flow mediated damage to endothelial cells, thereby slowing the progression of atherosclerotic plaques (Nollert *et al.*, 1992), and also lower the probability of developing an aneurysm at the abdominal aorta. Lower stress levels would also slow the rate of myointimal thickening in arterial grafts (Schwartz *el al.* 1992) and help moderate the increases in arterial blood pressure in hypertension.

Acknowledgements

H. R. C. gratefully acknowledges the National Institutes of Health (NIH Grant Number 5T32HD07417-04) for their support in funding this project. The authors also thank the Center for Applied Mathematics and Statistics at NJIT and UMDNJ for providing facilities for this work.

References

Asakura, T., and Karino, T. (1990). Flow patterns and spatial distribution of atherosclerotic lesions in human coronary arteries. Circ. Res.; 66: 1045 - 1066.

Bulanowski, E. A. and Yeh, H., (1971) Hemodynamic flow in anisotropic, viscoelastic thick-wall vessels. *J. Appl. Mech.* **38**, 351-362.

Chuong, C. J., and Fung, Y. C., (1986) On Residual Stresses in Arteries. *J. Biomech. Eng.* **108**, 189-192.

Chuong, C. J. and Fung Y. C., (1983) Three-Dimensional Stress Distribution in Arteries. *J. Biomech. Eng.* **105**, 268-274.

Demiray, H., and Vito, R. P., (1983) On large periodic motions of arteries. *J. Biomechanics* **16**, 643-648.

Fry, D. L., (1969) Certain histological and chemical responses of the vascular interface to acutely induced mechanical stress in the aorta of the dog. *Circ. Res.* **24**, 93-108.

Fung, Y. C., Liu, S. Q., and Zhou, J. B., (1993) Remodeling of the constitutive equation while a blood vessel remodels itself under stress *J. Biomech. Eng.* **115**, 453-459.

Guyton, A.C., and Hall, J.E. (1996). Textbook of Medical Physiology (9th ed.). pp. 244, 256-7. W.B. Saunders Co., Philadelphia, Pa.

Misra, J. C., and Chakravarty, S., (1982) Dynamic response of arterial walls in vivo. *J. Biomechanics* **15**, 317-324.

Misra, J. C., and Singh, S. I., (1984) Pulse wave velocities in the aorta. *Bull. Math. Biol.* **46**, 103-114.

Moore, J.E., Ku, D.N., Zarins, C.K., and Glagov, S. (1992). Pulsatile flow visualization in the abdominal aorta under differing physiological conditions: Implications for increased susceptibility to atherosclerosis. J. Biomed. Eng.; 114: 391 - 397.

Nichols, W. W., and O'Rourke, M. F. (1990). McDonald's Blood Flow in Arteries (3rd ed.) p. 399, Lea and Febiger, Philadelphia.

Nollert, M.U., Hall, E.R., Eskin, S.G., and McIntire, L.V. (1989). Effect of flow on arachidonic acid metabolism in human endothelial cells. Biochimica et Biophysica Acta; 1005: 72 - 78.

Nollert, M.U., Panaro, N.J., and McIntire, L.V. (1992). Regulation of genetic expression in shear stress - stimulated endothelial cells. Ann. N.Y. Acad. Sci.; 665: 94 - 104.

Okano, M., and Yoshida, Y. (1992). Endothelial cell morphometry of atherosclerotic lesions and flow profiles at aortic bifurcations in cholesterol fed rabbits. 114: 301 - 308.

Patel, D. J., and Fry, D. L., (1966) Longitudinal tethering of arteries in dogs. *Circ. Res.* **19**, 1011-1021.

Rogers, T. J., and Lee, E. H. (1964) The cylinder problem in viscoelastic stress analysis. *Quart. Appl. Math.* **22**, 117-131.

Schwartz, L.B., O'Donohue, M.K., and Purut, C.M.(1992). Myointimal thickening in experimental vein grafts is dependent on wall tension. J. Vasc. Surg.; 15: 176 - 186.

Simon, B. R., Kobayashi, A. S., Strandness, D. E., and Widerhielm, C. A., (1972), Re-evaluation of arterial constitutive laws. *Circ. Res.* **30**, 491-500.

Singh, S. I., and Devi, L. S., (1990) A study on large radial motion of arteries in vivo. *J. Biomechanics* **23**, 1087-1091.

Sobin, S. and Tremer, H. M., (1980) Cylindricity of the arterial tree in the dog and the cat. *Fed. Am. Soc. Exp. Bio.* **39**, 269.

Vorp, D. A., Rajagopal, K. R., Smolinski, P. J., and Borovetz, H. S., (1995) Identification of Elastic Properties of Homogeneous Orthotropic Vascular Segments in Distension. *J. Biomechanics* **28**, 501-512.

Figure Captions

Fig. 1 Cross-sectional representation of unloaded thoracic artery of the rabbit at the stress free reference state assuming residual stresses (State 0); for the unloaded intact artery (State 1); and for the subsequent loaded state under pressure (State 2).

Fig. 2 Circumferential stresses $t_{\theta\theta}$ through the deformed wall of the artery due to dynamic pressure at the inner wall, in the absence of external pressure, both in the residual and non-residual stress cases, at end diastole. The effect of taking into account the residual stresses is to decrease the very large circumferential stress at the inner wall by 66% and reduce the stress gradient at the inner wall by 94%.

Fig. 3 Pressure at the inner wall of the artery versus time in the absence of external pressure for both the non-residual stress case and the residual stress case with various frequencies. The dynamical pressure at the inner wall is the same in the presence or absence of residual stresses.

Fig. 4 Circumferential stress $t_{\theta\theta}$ at the inner wall of the artery versus time in the absence of external pressure in the non-residual stress case with various frequencies.

Fig. 5 Circumferential stress $t_{\theta\theta}$ at the inner wall of the artery versus time in the absence of external pressure in the residual stress case with various frequencies. From Figs. 4 and 5, we observe that the maximum stress of approximately 636 kPa in the non-residual stress case is reduced to 240 kPa in the residual case while the minimum stress is reduced from approximately 207 kPa to 86 kPa. These do not depend upon the heart rate.