**Reevaluation of Arterial Constitutive Relations**

**A FINITE-DEFORMATION APPROACH**

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**ABSTRACT**

The purpose of this investigation was to use the finite-deformation theory of elasticity to interpret pressure-diameter data for in situ canine aortas and other arterial response data reported in the literature. A meaningful mechanical property for arterial tissue was identified as \( \partial W_1/\partial I \), the partial derivative of the strain-energy density function with respect to the first strain invariant. An exponential function was found to characterize the mechanical property \( \partial W_1/\partial I \) for all arteries considered. Thin-walled tube stress approximations were found to result in inaccurate values for arterial stresses and incremental elastic mechanical properties. Wave speeds calculated using \( \partial W_1/\partial I \) for these arterial tissues agreed well with experimental measurements of wave speeds reported in the literature. Elevated values for strain-energy density were found in the inner arterial tissue layers. These high values for strain energy may contribute to atherogenesis in relatively straight arteries (e.g., the abdominal aorta) subjected to hypertension.

**KEY WORDS**

atherosclerosis strain-energy density wave speeds dogs

The age-associated disorders and diseases of the arterial system represent some of the most actively investigated current clinical and research problems. One such disorder—atherosclerosis—is thought to result in changes in arterial mechanical response. An investigation of possible relationships between atherosclerosis and arterial stiffening would necessarily require an accurate measure of in vivo mechanical properties (constitutive relations) of arterial tissue.

The determination of arterial mechanical properties has been the objective of researchers for more than a century; the earliest works were reported in 1808 and 1809 by Young (1, 2) and by Roy (3) in 1880. Recent studies of arterial mechanical properties have been based on measurements of arterial geometry and internal pressures. Incremental elastic properties have been determined using an isotropic model (4) and cylindrically orthotropic models (5–7) for a number of arterial tissues. Elastic properties have also been calculated using the Moens-Korteweg equation (Eq. 18 below) and measurements of the wave speed in arterial tissue (8, 9).

Both the Moens-Korteweg equation and the incremental stress-strain relations are based on a modified linear theory valid for "small deformations" (deformed and undeformed geometry approximately the same) of a "thick-walled" cylinder, i.e., uniform tangential stress in the wall. Experimental work by Arndt et al. (10) has shown that during the cardiac cycle the resulting in vivo deformation may be greater than 10% in terms of tangential strain. In addition, very large deformations...
(60–100% tangential strain) occur for pressures from 0–200 mm Hg (5). A finite-deformation analysis of the arterial cross section (11) with a thick-walled model allowing possible stress gradients in the arterial wall has shown that significant gradients in tangential stress may be present in arteries which have previously been assumed to be "thin-walled" cylinders in a modified linear elastic model. Hence, available elastic arterial constitutive relations should be reevaluated on the basis of a thick-walled model including finite deformations since the necessary analytical and numerical procedures (11) are now available.

Additionally, the strain-energy density function (determined below to characterize the arterial mechanical properties) could be a useful indicator in atherogenesis. Fry (12) has postulated that atherosclerosis may occur at locations in the arterial tree where the strain energy is abnormally increased, e.g., lesions commonly occur near arterial bifurcations. The strain energy based on the finite-deformation theory of elasticity (13) could be used to determine such abnormal levels of strain energy in the arterial wall.

The objectives of the present study were the following: (1) to determine the elastic mechanical properties for in situ canine abdominal aortas at varying axial stretch and for various other arteries considered in the literature (5, 14, 15) with the finite-deformation, thick-walled formulation previously developed (11); (2) to calculate the wave speed for these arterial tissues as a function of intraluminal pressure based on the finite-deformation mechanical properties and compare the results with experimental wave-speed data (8, 9); (3) to determine stress gradients present in the arterial cross sections considered with the above mechanical properties; (4) to quantify the distribution of strain energy associated with the stress and strain gradients in the arterial cross sections at normal and abnormally elevated intraluminal pressures.

**Theory**

Only the elastic mechanical properties were determined for arterial tissues. Experiments to characterize the history-dependent response in the presence of large deformations (e.g., for rubber materials) indicate that even for simple extension a complex model is needed to describe the response (16). The large-deformation elastic response (determined below) provides a portion of a history-dependent model. In this study, the effect of prolonged, abnormally elevated pressures on arterial tissue was of particular interest. Hence, a static analysis using the "elastic" response of arterial tissue was carried out by considering the arterial deformations and pressures measured only at times after initial stress-relaxation effects had disappeared.

An artery was also assumed to be an axisymmetric cylinder constrained at constant length (plane strain). Arterial tissue was considered to be a homogeneous, incompressible material. Directional mechanical response was assumed to be described by a transversely isotropic model, i.e., an artery was permitted a different mechanical response in the radial and axial directions or the tangential and axial directions, but was assumed to be isotropic in the cross-sectional plane. Supporting evidence for these assumptions has been previously reported (11).

The elastic mechanical properties for arterial tissue can be characterized by constitutive relations (stress-strain relations) based on strain energy, i.e., the amount of mechanical energy stored in the material due to deformation. This strain energy can be expressed as a function with magnitude equal to the energy required to deform a given volume of the material. This function is known as the strain-energy density function and will be denoted by W. If the form for W can be determined for a material, then the elastic mechanical response of that material can be quantified. The formulation and methods for determining the form of W for arterial tissue are presented briefly below.

In general, W is a function of the state of strain, hence

\[ W = W(\gamma_{11}, \gamma_{22}, \gamma_{33}, \gamma_{12}, \gamma_{23}, \gamma_{13}, \gamma_{12}, \gamma_{23}, \gamma_{31}, \gamma_{32}, \gamma_{33}) \]
where $\gamma_{n1}$, $\gamma_{n2}$, etc. are components of strain, e.g., $\gamma_{n1}$ and $\gamma_{n2}$ denote the radial and tangential components of strain, respectively. For axisymmetric plane strain of an incompressible material, the strain-energy density function can be written as a function of strain parameters (strain invariants) which are combinations of the strain components (13).

The particular strain invariant of interest for a transversely isotropic material is $I$ given by

$$I = 3 + 2(\gamma_{n1} + \gamma_{n2} + \gamma_{n3}).$$

Let $K$ denote the axial strain $\gamma_{n3}$, i.e.,

$$K = \gamma_{n3} = \frac{1}{2}(\lambda^2 - 1),$$

where $\lambda$ is the axial extension ratio (the ratio of deformed to undeformed arterial length), which is constant for plane strain. Under the above assumptions, the strain-energy density function is dependent only on $I$ and $K$ (13), and the function can be expressed as

$$W = W_1(I, K).$$

The deformation, $Q$, at a point in the arterial cross section is defined as the ratio of the undeformed radial coordinate, $r_1$, of the point to the deformed radial coordinate, $r$, of that point. Under the above assumptions, $Q$ can be written as

$$Q = \frac{r}{r_1} = \frac{\lambda^2 + C_0}{r},$$

where

$$C_0 = \frac{a_1^2}{\lambda} - r_1^2 = \frac{a_2^2}{\lambda} - r_2^2$$

and $a_1$ and $r_1$ are the undeformed and deformed external radii and $a_2$ and $r_2$ are the undeformed and deformed internal radii of the artery. The invariant $I$ may be expressed in terms of $Q$ and $\lambda$ as

$$I = \lambda^2 + \frac{Q^2}{\lambda^2} + \frac{1}{Q^2}. $$

The only nonzero stresses in the cross-sectional plane are the radial and tangential components given by

$$\tau^{11} = 2h + 2 \frac{\partial W_1}{\partial I} \left( \frac{Q^2}{\lambda^2} \right), \quad \text{(radial stress)} \quad (8a)$$

$$\tau^{22} = 2h + 2 \frac{\partial W_1}{\partial I} \left( \frac{1}{Q^2} \right), \quad \text{(tangential stress)} \quad (8b)$$

where the above stresses are measured per unit of deformed arterial cross section. Note that Eqs. 8a and 8b are analogous to the stress-strain relations of classical elasticity, e.g., for an isotropic incompressible material

$$\tau_{rr} = p + 2Ge_{rr}, \quad \text{(radial stress)} \quad (9a)$$

$$\tau_{\theta\theta} = p + 2Ge_{\theta\theta}, \quad \text{(tangential stress)} \quad (9b)$$

where $e_{rr}$ and $e_{\theta\theta}$ are linear radial and tangential strain components, respectively, $p$ is the hydrostatic pressure, and $G$ is the shear modulus of the material. Hence, the function $\partial W_1/\partial I$ is a mechanical property analogous to the constant $G$ of linear elasticity.

In the above, $h$ is a "hydrostatic pressure term" (in general, $2h \neq p$) which can be determined from equilibrium and incompressibility requirements to be of the form

$$h = -\frac{1}{2}L(r) - \frac{Q^2}{\lambda^2} \frac{\partial W_1}{\partial I} + H, \quad (10)$$

where

$$L(r) = 2 \int_{r_1}^{r} \frac{\partial W_1}{\partial I} \left( \frac{Q^2}{\lambda^2} - \frac{1}{Q^2} \right) \frac{dr}{r}. \quad (11)$$

If the artery is assumed to be subjected to internal pressure, $P$, and zero external pressure than $H = 0$ and

$$P = \tau^{11} \bigg|_{r = r_2} \quad (12)$$

or by Eqs. 8a, 8b, 10, and 11

$$P = 2 \int_{r_1}^{r} \frac{\partial W_1}{\partial I} \left( \frac{Q^2}{\lambda^2} - \frac{1}{Q^2} \right) \frac{dr}{r}. \quad (13)$$

The above represents an equilibrium condition for the artery, i.e., the pressure, $P$, is balanced by the summation of forces in the arterial wall (the integral from $r_1$ to $r_2$).

Eq. 13 can be better understood by comparing it to the classical elasticity solution for a pressurized thick-walled tube, i.e., using Eq. 9

$$P = \int_{r_1}^{r} (\tau_{\theta\theta} - \tau_{rr}) \frac{dr}{r} = 2 \int_{r_1}^{r} G(e_{\theta\theta} - e_{rr}) \frac{dr}{r}. \quad (14)$$
Again, $\vartheta W_1/\vartheta I$ and $G$ have analogous roles. For a linear material, $G$ is constant and the integration in Eq. 14 can be carried out with $G$ outside the integral, whereas, in Eq. 13, $\vartheta W_1/\vartheta I$ is a function which must be included when integrating from $r_1$ to $r_2$.

By Eq. 6 $r_1$ is dependent on $r_2$,

$$r_1 = \sqrt{r_2^2 + \frac{a_1^2 - a_2^2}{\lambda}},$$  

and $P$ is therefore a function of $r_2$, i.e.,

$$P = L(r_2).$$  

With these results, the wave speed, $c_w$, for a small amplitude pressure pulse passing down the artery can be determined by the method of characteristics (17) as

$$c_w = \sqrt{\frac{2}{2\rho_F} \frac{dP}{dr_2}} = \sqrt{\frac{2}{2\rho_F} \frac{dL}{dr_2}}$$

where $\rho_F$ is the density of the fluid (blood). For small deformation of a thin, linearly elastic, isotropic tube, the above can be reduced to the familiar Moens-Korteweg equation

$$C_w = \frac{E_t}{r_1},$$

where $t$ is the arterial wall thickness and $E$ the elastic modulus.

The strain energy may be written as a function of $r$ and $\lambda$,

$$W = W_0(r, \lambda) = W_1(I, K),$$

and hence

$$\frac{\partial W_0}{\partial r} = \frac{\partial W_1}{\partial I} \frac{\partial I}{\partial r} = \frac{2}{r} \left( \frac{Q^4 - \lambda^2}{\lambda Q^4} \right) \frac{\partial W_1}{\partial I}$$

$W_0(r, \lambda)$ is then given by

$$W_0(r, \lambda) = \int_{r_1}^{r} \frac{\partial W_0}{\partial r} dr + F(\lambda)$$

$$= \frac{2}{\lambda^2} \int_{r_1}^{r} \left[ \frac{(Q^4 - \lambda^2)}{Q^4} \right] dr + F(\lambda),$$

where $F(\lambda)$ is a constant at constant $\lambda$.

In the above relations if the mechanical property function, $\vartheta W_1/\vartheta I$, can be determined for arterial tissue, then the in-plane stresses, the wave speed, and the distribution of strain energy through the arterial wall can be quantified at constant $\lambda$.

**Methods**

The methods for the determination of $\vartheta W_1/\vartheta I$ (described in detail in ref. 11) are summarized briefly below. The required supporting experimental data necessary to determine $\vartheta W_1/\vartheta I$ are measurements of static internal pressures and corresponding external (or internal) radii over the desired pressure range, the in situ axial extension ratio, and the unstressed internal and external radii. In our experiments, these parameters were determined for in situ canine abdominal aortas. A stainless steel dumbbell-shaped cylinder (inserted through a longitudinal incision) was used to constrain an exposed aortic segment at constant axial extension ratio. For one test, the axial extension ratio was varied to obtain mechanical response data at several constant axial extensions, including the in situ value. The aortic segment was inflated to various constant volumes after initial inflations to minimize hysteresis (14). After initial stress relaxation, corresponding values of diameter and pressure were recorded using the apparatus previously described (11). The in situ axial extension was determined by measuring a gauge length on the arterial segment before and after excision. Unstressed dimensions were obtained from photographs of thin sections of the excised arterial cross section.

Several other investigators have conducted similar experiments in the past. Tickner and Sacks (5) measured pressures and resulting geometry changes for a large number of arteries. The curve-fit pressure-external radius data, corresponding axial extensions, and initial geometries for a canine thoracic aorta and a human brachial artery were used in this study to determine mechanical properties. Lee et al. (14) presented constant-length pressure-radius data for a number of arterial tissues. A canine carotid artery considered at two axial extensions was analyzed here. Additional experimental data were obtained from a study of the mesenteric arteriole of a frog.
TABLE 1

<table>
<thead>
<tr>
<th>Artery</th>
<th>( \lambda ) (X10^4 dyn/cm²)</th>
<th>C</th>
<th>k</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canine abdominal aorta 1</td>
<td>1.53</td>
<td>9.9</td>
<td>2.10</td>
</tr>
<tr>
<td>Canine abdominal aorta 2</td>
<td>1.28*</td>
<td>21.4</td>
<td>3.23</td>
</tr>
<tr>
<td>Canine abdominal aorta 2</td>
<td>1.48†</td>
<td>25.8</td>
<td>3.75</td>
</tr>
<tr>
<td>Canine abdominal aorta 2</td>
<td>1.68§</td>
<td>113</td>
<td>2.56</td>
</tr>
<tr>
<td>Canine thoracic aorta (5)</td>
<td>1.45</td>
<td>11.9</td>
<td>0.56</td>
</tr>
<tr>
<td>Human brachial artery (5)</td>
<td>1.20</td>
<td>0.98</td>
<td>11.8</td>
</tr>
<tr>
<td>Frog mesenteric arteriole, relaxed (15)</td>
<td>1.58†</td>
<td>2.98</td>
<td>0.60</td>
</tr>
</tbody>
</table>

\[ \frac{\partial W}{\partial I} = Ce^{(t-\lambda)} \] and \( I_0 = \lambda + 2/\lambda \). Number of vessels tested is given in parentheses.

*\( \lambda < \lambda \) in situ.
†\( \lambda = \lambda \) in situ.
§\( \lambda > \lambda \) in situ.
¶Assumed value.

For all arteries considered, the mechanical property function \( \partial W/\partial I \) was determined to be an exponential function of the form

\[ \frac{\partial W}{\partial I} = Ce^{(t-\lambda)} \] (22)

where \( I_0 \) is the value of \( I \) when \( P = 0 \) given by

\[ I_0 = \lambda^2 + \frac{2}{\lambda} \] (23)

and \( C \) and \( k \) are constants for a given axial extension. The form for \( \partial W/\partial I \) was determined in the following way (11): (1) solve Eqs. 8a and 8b for \( \partial W/\partial I \) in terms of \( \tau^{11}, \tau^{22}, Q, \) and \( \lambda \); (2) calculate approximate values for \( \partial W/\partial I \) using values of thin-walled tube stress for \( \tau^{11} \) and \( \tau^{22} \) (Eq. 29) at each pressure level; (3) estimate values for \( C \) and \( k \) from a semilog plot of approximate \( \partial W/\partial I \) vs. \( I_1 \); (4) substitute these values of \( C \) and \( k \) until the pressure-radius response predicted by Eq. 13 agrees with experimental results. Table 1 lists the final values for \( C \) and \( k \) determined for each artery considered.

Using these values for \( C \) and \( k \) and the theory outlined above, an analysis of the arterial deformation data was carried out to determine the in-plane stresses, \( \tau^{11} \) and \( \tau^{22} \); the wave speed, \( c_w \); and the strain-energy distribution, \( W_0(r, \lambda) \). The integrations indicated in the above formulas were performed numerically using Simpson’s rule. The differentiation of \( L(r_2) \) in Eq. 16 was accomplished using a finite-difference algorithm, i.e.,

\[ \frac{dL}{dr_2} = \frac{L(r_2 + \delta) - L(r_2)}{\delta}, \] (24)

where \( \delta \) is an arbitrarily small number.

**Results and Discussion**

The mechanical response for all arteries considered was characterized by the mechanical property function \( \partial W/\partial I \). An exponential form for \( \partial W/\partial I \) yielded pressure-radius response curves which agreed closely with experimentally measured pressure-radius data. Hence by Eqs. 11, 16, and 22 the arterial pressure-radius response is given by

\[ P = 2C \int_{r_1}^{r_2} e^{(t-\lambda)} \left( \frac{Q^2}{\lambda^2 - Q^2} \right) \frac{dr}{r^3}, \] (25)

with values for \( C \) and \( k \) listed in Table 1.

The mechanical properties for all arteries considered were compared by plotting the value of \( \partial W/\partial I \) at the average deformed radial coordinate (denoted by \( \frac{\partial W}{\partial I} \mid r_2 \)) vs. internal pressure, \( P \), as shown in Figure 1. Note that a similar plot for an incompressible rubber tube (Mooney material) would be a series of horizontal straight lines, since for a Mooney material the strain-energy density function is (13)

\[ W = W(I_1, I_2) = C_1(I_1 - 3) + C_2(I_2 - 3), \] (28)

where \( C_1 \) and \( C_2 \) are constants and \( I_1 \) and \( I_2 \) are strain invariants given by

\[ I_1 = I \]

and

\[ I_2 = \lambda^2 + \left( I - \lambda^2 \right) - \frac{1}{\lambda^2} \] (27)

for axisymmetric plane strain. Hence, \( \partial W/\partial I \) for a Mooney material is a constant at each \( \lambda \),
FIGURE 1
Comparison of arterial mechanical properties.

For the arteries considered in this study (excluding canine abdominal aorta 2 for \( \lambda = 1.68 \), which is greater than \( \lambda \) in situ) the plot of \( \frac{\partial W_A}{\partial I} \) vs. \( P \) is nearly linear. The positive slope of the curves in Figure 1 is indicative of the stiffening phenomenon observed in pressure-radius curves for arterial tissues. The results for \( \frac{\partial W_A}{\partial I} \) vs. \( P \) for canine abdominal aorta 2 and the canine carotid artery indicate that the mechanical property \( \frac{\partial W_A}{\partial I} \) is increased when the axial extension, \( \lambda \), increases (Fig. 1). The increase in \( \frac{\partial W_A}{\partial I} \) is due to the directional response of the arterial tissue and the thinning of the arterial cross section which accompanies increased axial extension. An isotropic material (no directional sensitivity in mechanical response) such as the Mooney material also exhibits increased \( \frac{\partial W_A}{\partial I} \) as \( \lambda \) is increased according to Eq. 28.

The radial stress, \( \tau^{rr} \), and the tangential stress, \( \tau^{r2} \), distributions were calculated for

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each artery. Significant stress gradients were determined in the arterial wall for all arteries considered. As internal pressure increases, these gradients in radial and tangential stress become increasingly steep. Figures 2 and 3 illustrate the tangential stress gradients at physiological mean pressure levels (P ≈ 100 mm Hg) and at abnormally elevated mean pressure levels (P ≈ 200 mm Hg). In Figures 2 and 3 normalized values for tangential stress, $S_{\theta}$, are presented as a function of percent of deformed thickness of the arterial cross section (%t). The thin-walled values for stress are given by

\[ \tau_{r\theta} = -\frac{P}{2} \quad \text{(thin-walled radial stress)} \] (29a)

\[ \tau_{\theta\theta} = \frac{Pf}{t} \cdot \quad \text{(thin-walled tangential stress)} \] (29b)

The normalized stress parameter of Figures 2 and 3 is defined by

\[ S_{\theta} = \frac{r^2 \tau_{r\theta}}{\tau_{\theta\theta}} \quad \text{(normalized tangential stress)} \] (30)

Figures 2 and 3 indicate the magnitude of the tangential stress gradients in the arterial wall. Similar gradients occur in values for the radial stress, $\tau_{rr}$. Even at the middle of the arterial wall (the average deformed radius where %t = 50%), significant differences between $\tau_{rr}$ and $r^2 \tau_{r\theta}$ may exist, i.e., $S_{\theta} \neq 1$ at %t = 50%. These differences become greater as pressure is increased until at hypertensive pressure levels significantly elevates values for $r^2 \tau_{r\theta}$ occur at the inner surface of the artery. At P ≈ 100 mm Hg and %t = 50%, the average percent errors in estimating stresses based on a thin-walled tube model were 118% for radial stress for all arteries and 46% for tangential stress. The average percent errors at P ≈ 200 mm Hg and %t = 50% were 180% and 70% for radial and tangential stresses, respectively.

The possibility of inaccuracy in values for arterial stresses implies that values for incremental elastic properties calculated using thin-walled modified linear theory (i.e., $\tau_{rr}$ and $\tau_{r\theta}$) may also be inaccurate. Consider for example the isotropic incremental modulus $E_{\text{inc}}$ (Eq. 33 below) often used as an arterial mechanical parameter. The relation for $E_{\text{inc}}$ is derived from

\[ E_{\text{inc}} = \frac{3(\Delta\sigma_{r\theta} - \Delta\sigma_{rr})}{2(\Delta\sigma_{\theta\theta} - \Delta\sigma_{rr})}, \quad \text{(31)} \]

where $\Delta\sigma_{rr}$ and $\Delta\sigma_{r\theta}$ are incremental radial and tangential stresses and $\Delta\sigma_{\theta\theta}$ and $\Delta\sigma_{rr}$ are incremental radial and tangential strains, respectively, for an initially pressurized incompressible, isotropic tube held at constant length. In the above, incompressibility requires that $\Delta e_{rr} = -\Delta e_{\theta\theta}$. At $r = r_1$, $\Delta e_{\theta\theta} = \Delta r_1/r_1$ and $\Delta \sigma_{rr} = 0$, hence

\[ E_{\text{inc}} = \frac{3r_1}{4} \left( \frac{\Delta\sigma_{r\theta}}{\Delta r_1} \right) \quad \text{(32)} \]

Note that when $\Delta\sigma_{r\theta}$ is approximated using a modified linear elasticity solution Eq. 32 reduces to the familiar equation

\[ E_{\text{inc}} = \frac{3}{2} \left( \frac{r_1^2}{r_1^2 - r_2^2} \right) \left( \frac{\Delta P}{\Delta r_1} \right) \quad \text{(33)} \]

However, when $\Delta\sigma_{r\theta}$ is set equal to $\Delta(r^2 \tau_{r\theta})$ in Eq. 33, then exact values for $E_{\text{inc}}$ are obtained. This calculation was carried out for canine abdominal aorta 1. The values for $E_{\text{inc}}$ obtained by Eq. 33 were compared to values for $E_{\text{inc}}$ using $\Delta(r^2 \tau_{r\theta})$ in Eq. 32 and the results were plotted as a function of pressure (Fig. 4). Values for $E_{\text{inc}}$ calculated using Eq. 33 were found to be overestimates of the actual values for $E_{\text{inc}}$ using $\Delta(r^2 \tau_{r\theta})$. The difference between these values for $E_{\text{inc}}$ increased as P increased, e.g., the difference in $E_{\text{inc}}$ was 89% at P = 100 mm Hg and 106% at P = 200 mm Hg. Even when values for $E_{\text{inc}}$ are compared at the average radius, a significant difference exists between modified linear theory and finite-deformation results for the arteries considered. This type of inaccuracy in values for $E_{\text{inc}}$ would also occur when the above approximations are used to calculate values for orthotropic incremental elastic properties. This inaccuracy is also present in the application of the Moens-Korteweg equation to determine E in Eq. 18 (incremental analysis).

A further illustration of the usefulness of the finite-deformation mechanical property, $\partial W_{\theta\theta}/\partial r_1$.
$\delta I$, is obtained by calculating the wave speed, $c_\omega$, in arterial tissue predicted by combining Eq. 25 with Eq. 17. The resulting values of $c_\omega$ are plotted as a function of $P$ as solid lines in Figure 5 for the canine aortas. The trend in the values for $c_\omega$ determined for the thoracic
aorta ($\lambda = 1.45$) agrees well with Anliker's (8, 9) experimentally measured values for $c_w$ (shown as data points in Fig. 5). The wave speeds in the abdominal aortas considered ($\lambda = 1.48, \lambda = 1.53$) were found to be higher than wave speeds in the thoracic aortas.

Finally, the strain-energy distribution $W_0$ ($r, \lambda$) as a function of percent arterial wall thickness ($\%t$) was determined using Eqs. 21 and 22. Figure 6 shows the strain-energy gradients (determined to within $F(\lambda) = $ constant) for all arteries considered at physiological and hypertensive pressure levels. In Figure 6 the strain energy due to pressurization is given by $\omega(r, \lambda) = W_0(r, \lambda) - F(\lambda)$. The values for strain energy become increasingly high for the inner arterial tissues. This effect is most pronounced at elevated internal pressures. The average percent increase in values for $\omega(r, \lambda)$ at the inner arterial surface was 150% for $P$ increasing from 100 to 200 mm Hg.

Freis (18) has pointed out that hypertension could be a causal factor in the formation of atherosclerotic lesions and does, at the outset, accelerate atherogenesis. Fry (12) has postulated that increased strain energy in the arterial wall may be a factor in atherogenesis. However, when linear concepts are used to estimate strain-energy levels, no explanation can be given for the occurrence of atherosclerosis in long, relatively straight arteries. The above increases in strain energy in the inner arterial tissue layers (calculated using finite-deformation theory) associated with abnormally elevated arterial pressure could provide a possible explanation (as a special case of Fry's hypothesis) for the atherosclerotic lesions so frequently observed in straight sections of the human abdominal aorta.

It is concluded that (1) The mechanical properties for arterial tissue can be quantified (using a thick-walled finite-deformation model) by $\frac{\partial W_1}{\partial l} = C e^{k(l-l_0)}$ for arteries held at constant length and subjected to internal pressures from 0 to hypertensive levels. (2) Formulas for stress in thin-walled tubes may result in significant errors in the values for radial and tangential arterial wall stresses even when these stresses are evaluated at the average deformed radial coordinate in the arterial cross section. (3) Values for incremental elastic mechanical parameters may be inaccurate if the calculation of these parameters is based on approximations of stress in thin-walled tubes. (4) The wave speeds predicted using arterial mechanical properties of the form $\frac{\partial W_1}{\partial l} = C e^{k(l-l_0)}$ compare closely with experimentally measured values for wave speeds (8, 9). (5) The strain energy, $W$, may reach significantly elevated values in the inner layers of the arterial wall, particularly at hypertensive levels of arterial pressure. If the
level of strain energy is a causal factor in the development of atherosclerosis as postulated by Fry (12), then gradients in W may contribute to the initiation of atherosclerosis in the inner layers of relatively straight arteries such as the abdominal aorta in a state of hypertension.

References
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