Biomechanical Study of the Constitutive Laws of Vascular Walls

In this study, the static mechanical behaviors of three different arterial walls are examined through the change in external radius due to distending pressure

by K. Hayashi, M. Sato, H. Handa and K. Moritake

ABSTRACT—Static mechanical behaviors of three different arterial walls were examined through changes in external radius due to distending pressure. In order to examine the distensibility of these vessels, distension ratio was defined as the ratio of eternal radius at each pressure to that at zero pressure. Linear relations were observed between the logarithmic pressure and the distension ratio, and they were described by an exponential function. Two parameters used in this equation were related quantitatively to the area fraction of elastin or collagen component occupied in the cross section of wall. Stress–strain relation was then determined from their pressure–diameter data by using finite-deformation theory. An exponential function was established between tangential stress and tangential strain. These results can be used to study the resistance of arterial walls to cardio-vascular disease.

Introduction

An understanding of cardio-vascular disease is very important because the number of deaths due to this disease has remarkably increased recently. There is an urgent need for understanding the mechanisms of atherosclerosis and those of initiation, growth and rupture of intracranial aneurysms. The disease is closely related to mechanical and hydrodynamical conditions imposed on vascular walls as well as to their structures.[1-3] Much can be learned, therefore, by studying these problems from the standpoint of biomechanics.

The mechanical properties of vascular walls are among the most important and the most fundamental factors of arterial resistance. In most studies, arterial walls have been assumed to be "thin-walled" cylinders in incrementally linear elastic models.[3-6] Infinitesimal-strain theory or the modified elastic theory normally cannot be used to adequately evaluate the large strains observed in vascular walls. Recently, some researchers have used the finite-deformation theory of elasticity to interpret the pressure–diameter data for vascular walls.[2,7]

Vascular walls consist mainly of three structural components such as elastin, collagen and smooth muscle. A complex interaction of these components governs the mechanical behaviors of bulk vascular walls. Each component is believed to have its unique and characteristic mechanical properties.[4,8] Therefore, mechanical properties of vascular walls must be considered in relation to their microscopic substructures.

In the present study, static mechanical behaviors of three different arterial walls were examined through the change in external radius due to distending pressure. The stress–strain relation of vascular walls was determined from their pressure–diameter data by using the finite-deformation theory. The parameters controlling the pressure–diameter relation or the stress–strain relation are quantitatively related to the volume or area fractions of their structural components.

Experimental Procedures

Figure 1 shows the experimental procedures. Segments of abdominal aorta, carotid artery and femoral artery excised from anesthetized mongrel dogs were used as test specimens. Portions were selected with no branches. These specimens were immersed in the physiological saline immediately after their excision and preserved in a refrigerator. The internal-pressure-loading tests were carried out within 48 h after sacrifice. All specimens were extended to their *in vivo* length during inflation. The arterial deformation and the pressure were measured after effects of initial stress relaxation had disappeared.

The testing apparatus consists of a pressure-loading part, a pressure-detecting part and a specimen-diameter-measuring part.[9] A schematic diagram is shown in Fig. 2. Internal pressure was measured through a pressure transducer attached at the end of an injection syringe. The output from the pressure transducer was recorded by an electromagnetic oscillograph after being amplified by a dynamic-strain meter. The tip of the syringe was set at the same site at which the specimen diameter was measured.

The specimen diameter was determined from the shadow area of the specimen. The reduced light flux, due to shadowing by the specimen, was measured by a silicon light sensor through a receiving slit. Its output was amplified and recorded simultaneously with

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the pressure output. Although a vascular wall is substantially noncircular in cross section, the measuring error of its diameter was about ±0.125 mm. A detailed description of the apparatus appears elsewhere.[9]

After static pressure–diameter tests, the specimen was immersed in a fixative solution[10] (10% formalin and 1% $CaCl_2$) under the constant pressure of 100 mmHg at 37° C for 2 hr for fixation. The fixed specimen was embedded in paraffin and then sectioned to about 10 microns in thickness by a microtome. Adjacent three slices from each specimen whose locations coincided with the illumination of light flux were examined by different staining methods. Resorcin-fuchsin was used to stain only elastin fibers among three structural components of vascular walls. Hematoxylin-picrofuchusin was used to stain only collagen fibers. Smooth muscle was stained with acid alizarin blue. The microstructure of each slice thus stained was photographed. Some examples are shown in Fig. 3. The profiles of the brightness distribution are shown at the bottom of this figure. They were obtained through a microphotometer by scanning each negative film in the radial direction. The area fraction occupied by each structural component in the bulk cross section was calculated by the following equation:[9]

$$A = \Sigma l_i / l_t \times 100(\%). \qquad (1)$$

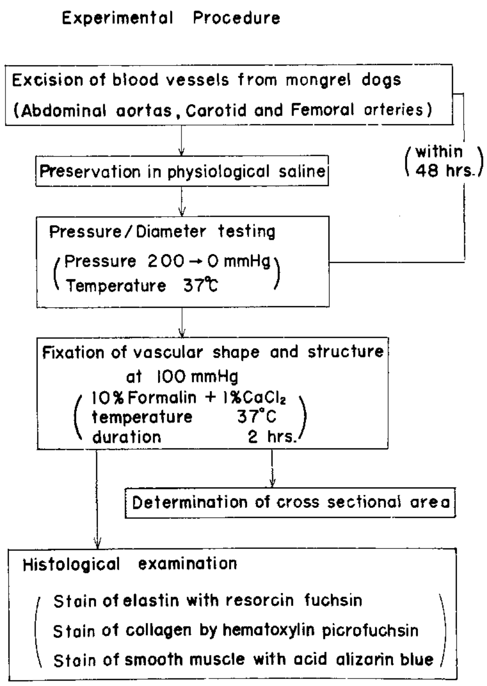

Fig. 1—Experimental procedures

Change in External Radius Due to Distending Pressure

Relations between the intraluminal pressures and the external radii of different three vessels are shown in Fig. 4. In the low-pressure range below 50 mmHg, wall radius increases rapidly with pressure. However, vascular walls become stiffer as they are distended in the pressure range between 50 and 150 mmHg. Above 150 mmHg, they almost lose their distensibility. In order to examine the distensibility of these vessels, the distension ratio, $(\lambda_\theta)_o$, was defined as the ratio of external radius at each pressure, R_o, to that at zero pressure, $(R_o)_{P=0}$.[11]

In Fig. 5, the distension ratio is plotted against the logarithmic pressure in the range of physiological pressure. Linear relations are observed between

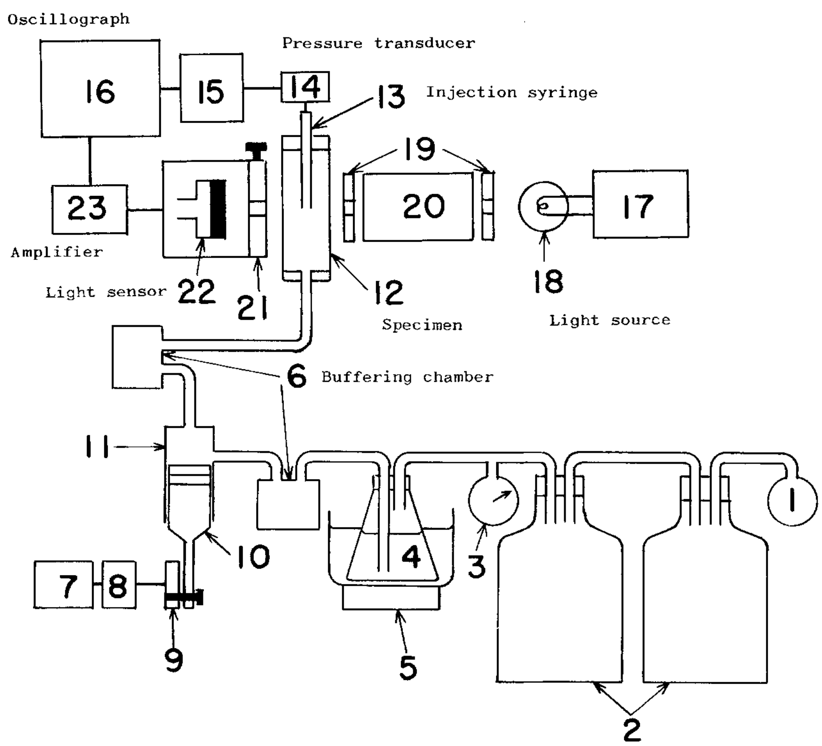

Fig. 2—Schematic diagram of testing apparatus

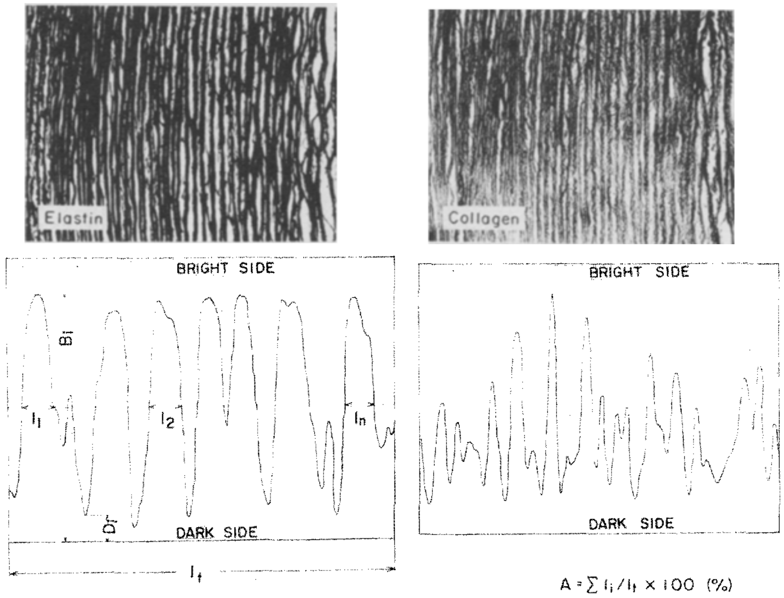

Fig. 3—Stained microstructures and distribution of brightness

these two values, which are described by the following general equation:

$$\log P = a + b\ (\lambda_\theta)_o \qquad (2)$$

Parameters a and b are used to represent the distensibility or stiffness of vascular walls. As seen in this figure, the distensibility of vascular walls depends upon the material. At the same internal pressure, abdominal aorta gave the highest distension ratio, carotid artery gave the medium, and femoral artery gave the lowest. This rank order of distensibility corresponds to that of the external radius of the vessel. It has been pointed out that elastin in a vascular wall decreases, while collagen increases, as its external radius decreases.[11,12] Since these three structural components have different mechanical properties, differences in their volume fractions for various specimens should have a great effect on their distensibility. In order to relate these mechanical behaviors with histological observations, parameters a and b were plotted against the area fraction of elastin and collagen.

Figure 6 shows these results as well as other data obtained by the present authors. Approximate linear relations are observed between the parameters a and b and the area fraction of elastin and collagen. These results are in agreement with the widely known results that elastin has high deformability, while collagen is a stiff component.[4,8] Using these data, relations between the internal pressure, P, and distension ratio, $(\lambda_\theta)_o$, are formalized in the following equations:

$$\log P = (0.18A_e - 8.10) + (-0.17A_e + 8.80)\ (\lambda_\theta)_o, \quad (3)$$

and

$$\log P = (-0.16A_c + 2.20) + (0.14A_c - 0.70)\ (\lambda_\theta)_o. \quad (4)$$

If these equations are applied to some other vascular walls, such as itracranial blood vessels whose distensibility or stiffness is difficult to determine experimentally, it will be possible to estimate their deformability from their histology.

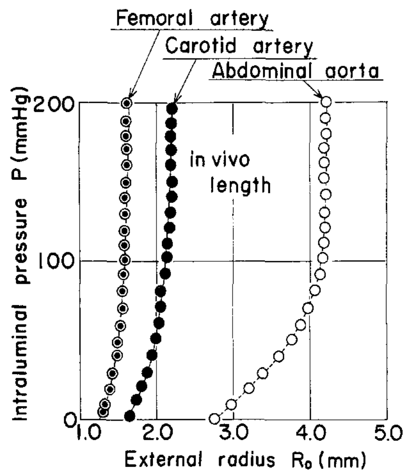

Fig. 4—Change in external radii of three vessels due to distending pressure

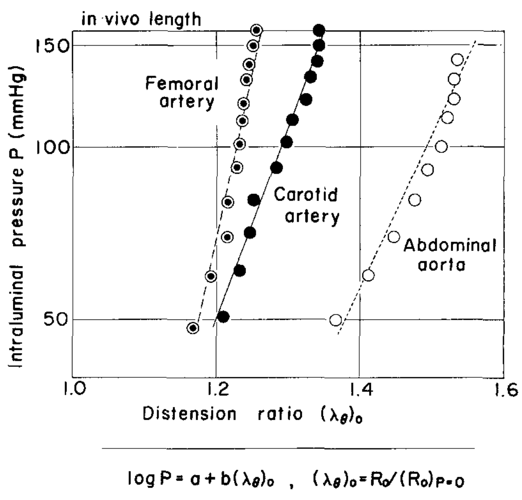

Fig. 5—Relations between intraluminal pressure and distension ratio

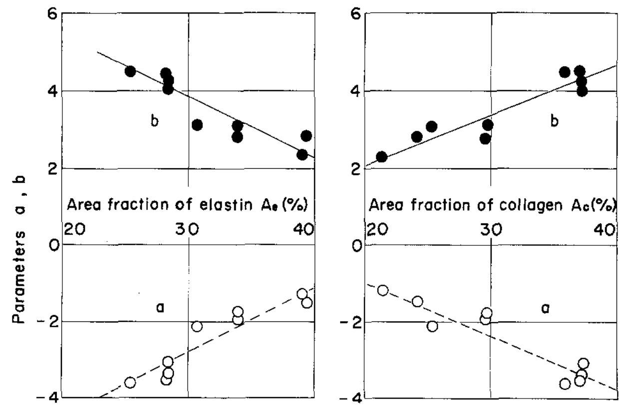

Fig. 6—Relations between parameters a and b and area fraction of elastin or collagen

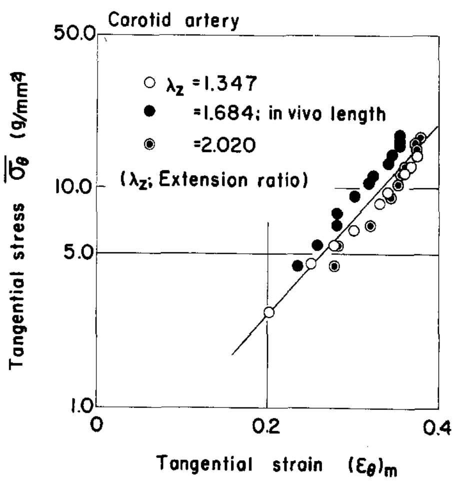

Fig. 7—Relation between thin-walled tube stress and mean strain

As for smooth muscle, such relations as shown in Fig. 6 could not be obtained. This is due to the fact that smooth muscle has almost no role on the elastic mechanical behaviors of vessels because of its viscoelastic property.[8]

Stress–Strain Relation of Vascular Wall

Finite-deformation theory was used to determine the stress–strain relation of vascular walls from their pressure–diameter data. Static analysis of elastic deformation was carried out for an axisymmetric cylinder constrained at a constant length. The artery was assumed to be a homogeneous and incompressible material and also to be isotropic in the cross-sectional plane.

A circular cylindrical tube, whose length is l in the unstrained condition and which has external and internal radii r_o and r_i, respectively, was considered. The tube was assumed strained in the following sequence of deformation after Green and Zerna:[13]

(1) a uniform simple extension in which its length becomes $l\lambda_z$, where λ_z is the extension ratio;

(2) a uniform inflation in which its length remains constant and its external and internal radii change to R_o and R_i, respectively.

The physical components of the strain in Lagrangean coordinates are obtained as follows;

$$\epsilon_{rr} = -\frac{1}{2\lambda_z}\left(\frac{\lambda_z R_o^2 - r_o^2}{\lambda_z R^2} + \lambda_z - 1\right), \tag{5}$$

$$\epsilon_{\theta\theta} = \frac{1}{2\lambda_z}\left[\frac{\lambda_z R_o^2 - r_o^2}{\lambda_z R^2 - (\lambda_z R_o^2 - r_o^2)} - \lambda_z + 1\right], \tag{6}$$

$$\epsilon_{zz} = \frac{1}{2}(\lambda_z^2 - 1). \tag{7}$$

The stress–strain relation of arterial tissues was determined as an exponential function given in the following equation:

$$\sigma_{\theta\theta} = k\, e^{n\epsilon_{\theta\theta}}. \tag{8}$$

This expression was estimated from the experimental results shown in Fig. 7, which is an example of the relation between the logarithmic tangential stress calculated by using the thin-walled theory and the mean tangential strain. Approximate linear relationship is observed between these two values and is described as follows:

$$\bar{\sigma}_\theta = k\, e^{n(\epsilon_\theta)_m}, \tag{9}$$

where

$$\bar{\sigma}_\theta = P R_i/t \quad (t = \text{wall thickness}), \tag{10}$$

$$(\epsilon_\theta)_m = \Sigma(\Delta R_m/R_m), \tag{11}$$

and

$$R_m = (R_o + R_i)/2. \tag{12}$$

On the basis of this relation, the form of stress–strain relation given by eq (8) was assumed for arterial walls. In the following, this assumption will be elucidated and these two material coefficients, n and k, will be determined.

As the materials are assumed to be homogeneous and isotropic, the equations of equilibrium are given as follows:

$$\frac{\partial\sigma_{rr}}{\partial R} + \frac{\sigma_{rr} - \sigma_{\theta\theta}}{R} = 0, \tag{13}$$

$$\frac{\partial\sigma_{\theta\theta}}{\partial\theta} = \frac{\partial\sigma_{zz}}{\partial Z} = 0. \tag{14}$$

After combining eqs (6), (8) and (13), integration gives the following equation:

$$\sigma_{rr} = \frac{k}{R}\int_{R_o}^{R} \exp\frac{n}{2\lambda_z}\left[\frac{\lambda_z R_o^2 - r_o^2}{\lambda_z R^2 - (\lambda_z R_o^2 - r_o^2)} - \lambda_z + 1\right] dR + C, \tag{15}$$

where C is an integral constant. As an artery is subjected to internal pressure P and zero external pressure, eq (15) finally becomes:

$$P = \frac{k}{R_i}\int_{R_i}^{R_o} \exp\frac{n}{2\lambda_z}\left[\frac{\lambda_z R_o^2 - r_o^2}{\lambda_z R^2 - (\lambda_z R_o^2 - r_o^2)} - \lambda_z + 1\right] dR. \tag{16}$$

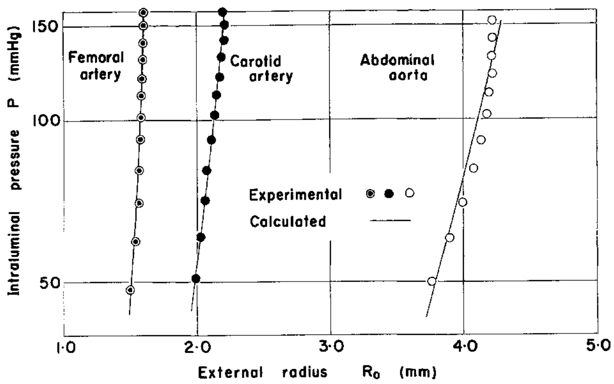

Fig. 8—Comparison of calculated results with experimental results

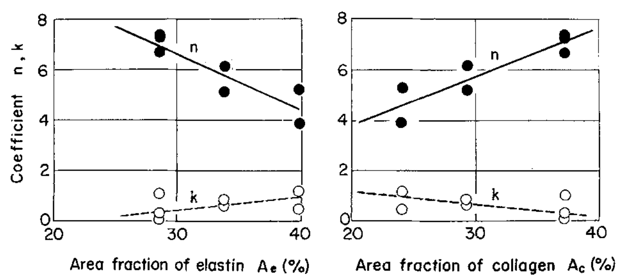

Fig. 9—Effect of histology on the coefficients of n and k

This equation represents an equilibrium condition for an artery inflated by the intraluminal pressure. Values of n and k must be determined to satisfy this final equation.

Determination of values of n and k was carried out as follows. First, approximate values of n and k were estimated by using eq (9) from a semilogarithmic plot of the thin-walled tube stress against the mean strain as shown in Fig. 7. The values of n and k thus obtained were substituted into eq (16). The calculated pressures, P, were plotted against the external radii, R_0, for comparison with experimental values. The steps mentioned above were repeated for various modified values of n and k until the resulting pressure–radius relation agrees with the experimental result. Figure 8 shows some examples of final results. Calculated values are observed to agree well with experimental results, which indicates the appropriateness of the theoretical procedures used in this study.

Variations in coefficients n and k with histology are shown in Fig. 9. Nearly linear relations are again observed between these coefficients and area fractions of elastin and collagen. These data show that the stress–strain relation of vascular walls can be determined explicitly from histology alone. These results can thus be used to study the resistance of arterial walls to cardio-vascular diseases.

Summary

Static mechanical behaviors of three different arterial walls were examined through the change in external radius due to distending pressure. In the low-pressure range below 50 mmHg, wall radius increased rapidly with pressure. However, vascular walls became stiffer as they were distended in the pressure range between 50 mmHg and 150 mmHg. Above 150 mmHg, much of the distensibility was lost.

Linear relations were observed between the logarithmic pressure, P, and the distension ratio, $(\lambda_\theta)_o$, and can be represented by the following equation:

$$\log P = a + b\,(\lambda_\theta)_o\,,$$

where parameters a and b are used to represent the distensibility of vascular walls.

Interaction between elastin, collagen and smooth muscle in vascular walls is considered to govern the mechanical behaviors of bulk vascular walls. Mechanical properties are then related to histological observations by a linear relation between parameters a and b and area fractions of elastin and collagen occupied in the bulk-wall cross section.

The stress–strain relation of vascular walls was determined from their pressure–diameter data by using the finite-deformation theory. This relation is:

$$\sigma_{\theta\theta} = k\, e^{n\epsilon_{\theta\theta}}\,.$$

Again, nearly linear relations were observed between the coefficients n and k and area fractions of elastin and collagen.

These results could be used to study the resistance of arterial walls to cardio-vascular disease.

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