§ 1. The correct interpretation of the parameters of pulse wave propagation is very important for the nontraumatic evaluation of the mechanical properties of blood vessels, which accounts for the interest in this question. Detailed literature on theoretical and experimental aspects of this question may be found in reviews by McDonald [1], Fung [2], and Anliker [3]. In considering pulse wave propagation as a problem in applied mechanics, the correct selection and basis of the theoretical model, for which suitable principles and equations of compact medium mechanics must be used, are of major importance. Since a model correctly reflects the significant aspects of the object studied, the results obtained and conclusions may be used for a correct evaluation of the mechanical properties of blood vessels on the basis of the wave characteristics of the propagation of arterial pressure measured in vivo.

From the viewpoint of geometry, the major arteries are considered as cylindrical thick-walled or even thin-walled tubes. The blood which flows through these arteries may be considered as a Newtonian liquid which is subject to laminar flow. The correct modelling of the mechanical behavior of the arterial walls is much harder in light of their complex structure; this leads to significant mathematical difficulties in solving the problem of pulse wave propagation. The many studies of the rheology of blood vessel tissue have shown that this tissue manifests pronounced physical nonlinearity, high deformability, and differing mechanical properties in the lengthwise, radial, and circumferential directions. There is a well-founded basis for considering that the wall material is incompressible and elastic. The mechanical behavior of blood vessels is complicated also as a result of the presence of smooth muscle fibers, which may contract upon the action of neurogenic and humoral factors, in the structure of the wall. This leads to changes of the strained and stressed states of the vessel and of the mechanical properties of the vessel tissue. In the physiological state, the radius of arterial vessels under the action of average arterial pressure is 170% greater than the radius in the unstrained state and the length under the effect of the surrounding tissues is 150% greater.

Thus, the problem of the stressed and strained states of blood vessels in situ has pronounced geometric nonlinearity. Various workers [4–9] have treated the effect of initial stresses on the wave propagation parameters. However, the change in the vessel dimensions and mechanical properties of the wall which must be followed for geometrical and physical nonlinearity were not taken into account in the parametrical study of the effect of initial stresses. Thus, the theoretical results obtained do not always agree with the experimental data. The theoretical feasibility of a more exact consideration of initial stresses was shown in our earlier work [10], in which equations of phase rates were derived using the theory of small strains applied on larger strains. However, the assumption of the isotropic nature of the material made in our earlier work [10] significantly limits the applicability of the results obtained for the qualitative and quantitative analysis of the dynamic behavior of real vessels.

The present author is unaware of a mechanical model which accounts for the effect of muscle activity on pulse wave propagation. The limited number of in vivo experiments are often subjected to conflicting interpretation [3, 11, 12].

In the present work, a model is proposed which more exactly accounts for the effect of initial stresses and the effect of muscle contraction on pulse wave propagation. This model is based on the following assumptions:

1) The pulse wave propagation process is considered axially symmetric and characterized by small amplitudes of displacements of the vessel wall from its final strained state and small amplitudes of liquid velocity relative to its steady-state velocity. This assumption is related to the relatively small changes in blood pressure over the cardiac cycle. The wavelength is taken as much greater than the arterial radius, which is in accord with the experimental data under physiological conditions [13].
2) The circulating blood is considered an incompressible Newtonian liquid subject to laminar flow.

3) The artery is considered as a thick-walled shell consisting of a substance which is incompressible, elastic, nonlinear, and curvilinear-orthotropic. Under the effect of mean arterial pressure and the surrounding tissues, the shell has finite strains in the axial and circumferential directions. The activation of smooth muscle causes an additional finite strain of the vessel in the circumferential direction due to tension in the muscle fibers. The predominant muscular effect in the circumferential direction is a result of the morphological structure of the blood vessel wall [14] and the assumption of a finite strain due to muscle contraction is based on the correct interpretation of the experimental data on the active behavior of arteries in vivo. This is explained by the fact that the effect of the stimulating action is not limited only by the arteries, but also leads to a contraction of the fine precapillary vessels, the major component of which is smooth muscle. As a result, the peripheral resistance increases, leading to an increase in arterial pressure and passive stretching of the arteries. This stretching is neutralized to a certain extent by arterial contraction caused by smooth muscle. However, if the transmural pressure is held constant, a 30% decrease in the arterial diameter is observed in the presence of vasoactive substances [15].

It is assumed that the stimulation of muscle caused by factors external to it is such that feedback between strains and muscle activity does not exist. This assumption is valid for large and medium arteries. The mechanical behavior of stimulated tissue is determined on the basis of its passive behavior (in the absence of stimulation) taking into account additional strain as a consequence of muscle contraction and muscle tension [16].

In determining the stressed and strained states of an artery under the action of mean pressure as well as during wave propagation, the artery is considered a membrane. Flexural rigidity may be ignored due to: 1) the existence of large strains; 2) the fact that the pulse waves have low frequencies under physiological conditions.

§2. In the hypothetical initial state $B_0$, in the absence of transmural pressure, axial forces, and an activating factor, let the artery have length $L_0$, mean surface radius $R_0$, and wall thickness $h_0$. Under physiological conditions in situ and under the effect of mean pressure $p$ only, the membrane is in state $B$ and has the following dimensions:

$$L = \lambda_1 L_0; \quad R = \lambda_2 R_0; \quad h = \lambda_3 h_0,$$

where $\lambda_1$, $\lambda_2$, and $\lambda_3$ are the major stretchings in the axial, circumferential, and radial directions, respectively. From the condition of incompressibility, it follows that $\lambda_1 \lambda_2 \lambda_3 = 1$ or $\lambda_3 = \frac{1}{\lambda_1 \lambda_2}$.

Under the action of a given external stimulus with maintenance of constant pressure $p$ and length $L$, smooth muscle contracts and the vessel converts to state $B^*$ with dimensions

$$R^* = \mu R = \mu \lambda_2 R_0; \quad h^* = \frac{h}{\mu} = \frac{h_0}{\mu \lambda_1 \lambda_2},$$

where $\mu$ is the geometrical index of vascular contraction in going from state $B$ to state $B^*$.

A coordinate system $\theta_\alpha$ ($\alpha = 1, 2$) related to the averaged surface of the membrane in state $B_0$ (Fig. 1a) is introduced to obtain tensor values of the strain and $\theta_3$ is measured relative to the direction of the external normal at every surface point. For metric tensors of a thin-walled membrane in state $B_0$, we have

$$a_{\alpha \beta} = g^{\alpha \beta} = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}; \quad g_{33} = g^{33} = 0; \quad g_{33} = g^{33} = 1 \quad (\alpha, \beta = 1, 2).$$

After two finite strains determined relative to (1) and (2), the covariant and contravariant components of the metric tensor in state $B^*$ have the following form: