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Pressure wave propagation in human arterial model -Comparative study of 1-D numerical simulation and experiment-

ヒト血管モデル内の圧力波伝搬
－１次元数値シミュレーションと実験の比較研究－

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1. Motivation

Evaluation of the pulse wave is effective for screening arteriosclerosis. In the previous study, we have verified that waveforms of the pulse waves changed markedly due to the arterial stiffness [1]. However, the pulse wave consists of two components, incident wave and multi-reflected waves. Clarification of complicated propagation phenomenon of these waves is important for understanding the nature of pulse wave in vivo.

In this study, we attempted to build a reliable one-dimensional numerical model for simulating wave propagation in human arteries. To evaluate the validity of the model, we compared the theoretical estimations with measured data obtained from the experiment using an artificial human artery model.

2. Modelization

The blood is considered to be viscous and incompressible fluid that flows through distensible tube vessels with circular cross section. In addition, the pressure wave length is so long compared to the vessel diameter that the flow is assumed as one-dimensional. Considering these hypotheses, the governing equations are formulated from integration of the Navier-Stokes equation and the equation of continuity:

Conservation of mass

$$\frac{\partial A}{\partial t}+\frac{\partial Q}{\partial x}=0 \qquad (1)$$

Momentum equation

$$\frac{\partial Q}{\partial t}+\frac{4}{3}\frac{\partial}{\partial x}\left(\frac{Q^2}{A}\right)=-\frac{A}{\rho}\frac{\partial p}{\partial x}-\frac{8\nu Q}{R^2} \qquad (2)$$

where Q is average flux over the cross section A. ρ and ν are fluid density and dynamic coefficient of viscosity. The pressure p is represented by a simple phenomenological nonlinearity and an attenuation term of Kelvin-Voigt Model:

$$p=\frac{E}{1-\sigma^2}\frac{h}{R^2}\left[\left(R-R_0\right)+\varepsilon\left(R-R_0\right)^2\right]+\eta\frac{\partial R}{\partial t} \qquad (3)$$

where E is the elastic modulus, h the wall thickness, σ the Poisson's ratio, R the tube radius, ε a parameter of nonlinearity, and η the viscosity, respectively.

3. Methods

Human artery model

Figure 1 shows the structure of a human arterial model. The configuration of each tube is shown in Table 1. Distensible polyurethane tubes were used for each artery and their tensile modulus was about 185 kPa. Silicone tubes (30 m) were connected at the end of each tube. The diameter, thickness, and elasticity of the silicone tubes were 8 mm, 2 mm, and 2.5 MPa, respectively. The elasticitic moduli of the silicone tubes were much higher than those of polyurethane tubes. The reflections of the flows arise at these connection points owing to the impedance mismatch, and the reflection coefficient at each point was estimated about 0.50 from the preliminary experiment [2].

Pressure and flow velocity measurement

An experimental system used was constructed with a pump and the arterial model. A pulsatile flow was input into the constructed arterial model from the heart using the pump. The input flow was a half-cycle of sinusoidal wave with the ejection time of 0.3 s and the total flow volume was 4.5 ml. Then, the inner pressure and flow velocity at the carotid artery were measured. The measurement point was set at 150 mm from the second bifurcation to point A (virtual cephalic vascular bed). A pressure sensor (Keyence, AP-10S) was used to measure the inner pressure wave, while an ultrasonic Doppler system (Toshiba Medical Systems, Aplio SSA- 700A) was used to measure the flow velocity. The center frequency of the ultrasonic pulse used (Toshiba Medical Systems, Probe PLT-1204AT) was 12 MHz.

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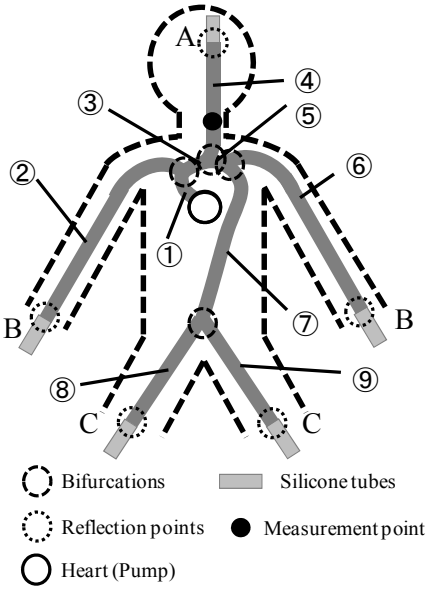

Fig. 1 Structure of an arterial network.

Table 1: Details of the human arterial model.

	Name	Length	Diameter	Thickness
①	Aorta arch A	35	12	2
②	R.subclavian radial artery	800	6	1.5
③	Aorta arch B	20	11	2
④	L.carotid artery	675	6	1.5
⑤	Aorta arch C	40	10	2
⑥	L.subclavian radial artery	710	6	1.5
⑦	Aorta	470	8	1.5
⑧	R.femoral artery	365	6	1.5
⑨	L.femoral artery	365	6	1.5

Unit in mm

Computation

Theoretical flow dynamics in numerical arterial model with the same configurations were computed by MacCormack method. Optimum parameters were decided by changing unknown values E_0, η, and ε. Here, to examine qualities of theoretical waves, we calculated dispersions of the difference between the measured and theoretical waves by using cost functions. Finally, the combination of parameters with the smallest dispersion was adopted.

4. Results and Discussion

Figures 2 and 3 show comparisons between the experimental and theoretical pressure waves and flow velocities. The coefficients determined by the cost functions were E_0 = 227 kPa, η = 0.036, and ε = 0.010. As a result, the tendency of the theoretical pressure wave fits closely with that of the measured wave. However, the amplitude difference between the theoretical and experimental waves became slightly larger in the latter part (less than 10% of the maximum amplitude). The difference was possibly caused by approximation error of integrated model, especially from viscous resistance of fluid. As for the comparison of flow velocity waveforms, the amplitude of the estimated velocity became smaller than that of the measured wave. Possible explanations for the difference were owing to the experimental problems and the scattering of ultrasonic beam. However, the comparatively small differences of theoretical and experimental waves prove the validity of 1-D modelization.

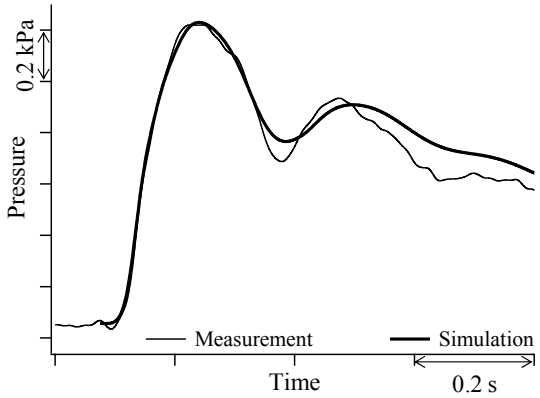

Fig. 2 Pressure waves as a function of time.

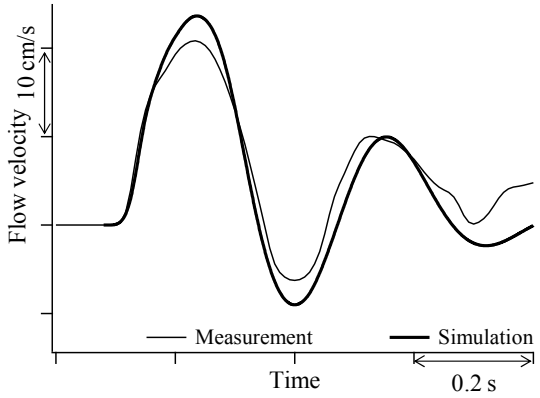

Fig. 3 Flow velocities as a function of time.

5. Conclusion

The 1-D modeling for simulating flow dynamics in the arterial model was formulated and its applicability was also validated with experimental results. In consequence, the theoretical results fit well with the experimental results when the parameters were determined properly. Thus, we can conclude that our modelization is acceptable and useful for simulating flow dynamics in the systemic arterial tree to understand effects of arteriosclerosis.

References

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2. Y. Yamamoto *et al.*, Jpn. J. Appl. Phys. **50** (2011) 07HF12.