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Accurate Ultrasonic Measurement of Viscoelasticity of Radial Arterial Wall with Correction of Change in Pulse Wave Velocity due to FMD Reaction

FMD 反応時の脈波伝播速度変化を補正した橈骨動脈壁粘弾性の高精度超音波計測

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1. Introduction

Cardiovascular disease has been cited as the top cause of death in Japan, and the primary cause of such diseases is atherosclerosis. The initial step of athersclerosis is considered to be the endothelial dysfunction[1)]. Although assessments of the endothelial function and viscoelastic properties of intima-media region are important for diagnosis of early-stage atherosclerosis, regional viscoelasticity has not yet been measured *in vivo*. In our group, a method for measurement of the transient change in viscoelasticity during flow-mediated dilation (FMD) was developed[2)]. However, in this method, the stress (blood pressure) and strain of the intima-media region are measured in different arms, and the change in pulse wave velocity due to FMD has not been considered[3)]. In the present study, we measured blood pressure waveforms with two pressure sensors to estimate the pulse wave velocity between the sensors. From the results, we corrected the pulse wave propagation time from the pressure sensor to the position of the ultrasound probe, and we successfully estimated viscoelasticity from the stress-strain relationship.

2. Principle

2.1 Measurement of stress-strain and regional pulse wave velocity

The blood pressure (stress) waveforms at two points were measured using two pressure sensors placed at a distance of 74 mm on the left radial artery. **Figure 1** shows the measured blood pressure waveforms at the radial artery. Time delay between the waveforms can be observed in Fig. 1. We estimated the pulse wave velocity by estimating the time delay using the cross-correlation function between the blood pressure waveforms and corrected the propagation time from the pressure sensor to the ultrasound probe. We used the waveform measured by the distal sensor to estimate the viscoelasticity.

The change in thickness (strain) of the radial arterial wall was measured using a 22-MHz ultrasound probe and applying the *phased-tracking method* to the received RF signals[4)].

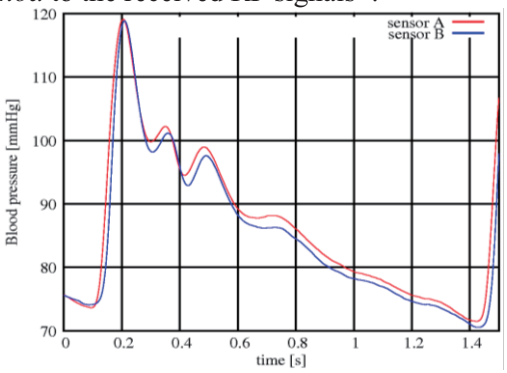

Fig. 1 Blood pressure waveforms obtained by the proximal and distal sensors (red and blue lines, respectively).

2.2 Viscoelasticity estimation

Let us assume that the viscoelastic properties of the intima-media region are modeled by the Voigt model. We estimate the elasticity and viscosity from stress-strain relationship by modeling the stress $\tau(t)$ as

$$\hat{\tau}(t) = E_S \int \mathrm{LPF}[\dot{\gamma}(t)]dt + \eta \mathrm{LPF}[\dot{\gamma}(t)] + \tau_0, \quad (1)$$

where $\hat{\tau}(t)$ is the modeled stress and $\dot{\gamma}(t)$, E_s, η, τ_0 and LPF[·] show strain rate, static elasticity, viscosity coefficient, bias stress corresponding to diastolic pressure, and low-pass filter, respectively.

2.3 *In vivo* measurement of FMD reaction

In this study, we placed cuffs on the upper arm instead of the forearm to reduce the influence of motion due to the increment of pressure of the cuff. For the measurement of the radial artery, ultrasonic RF echoes (transmit center frequency: 22 MHz) and blood pressure waveforms were acquired at sampling frequencies of 66.5 MHz and 10.3 kHz, respectively. This acquisition was repeated for 2 minutes at rest before avascularization and for 3 minutes after recirculation.

3. Results

Figure 2 shows the measured stress-strain relationship of the radial arterial wall with and without correction of the change in pulse wave velocity. By considering the propagation time, the directions of hysteresis loops were corrected from counterclockwise to clockwise.

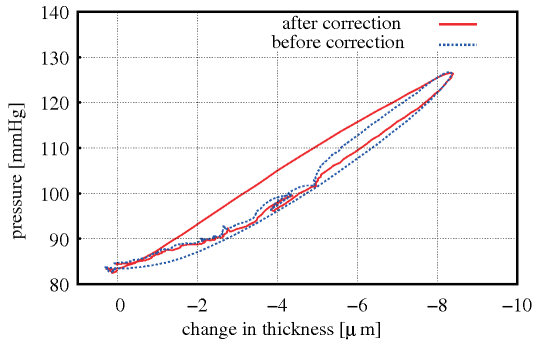

Fig. 2 Measured stress-strain relationships with and without correction of the pulse wave velocity (red and blue lines, respectively).

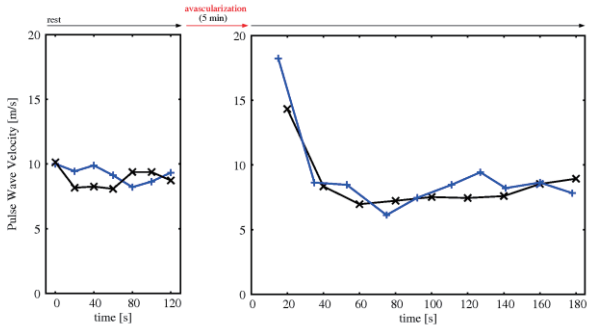

Fig. 3 Transient change in the pulse wave velocity estimates when avascularization was at the upper arm (black) and the forearm (blue).

The blue and black lines in **Fig. 3** show the transient changes in the pulse wave velocities measured at upper arm and forearm, respectively. Just after avascularization, the pulse wave velocity was very high and, then, it returns to the pulse wave velocities similar to those at rest.

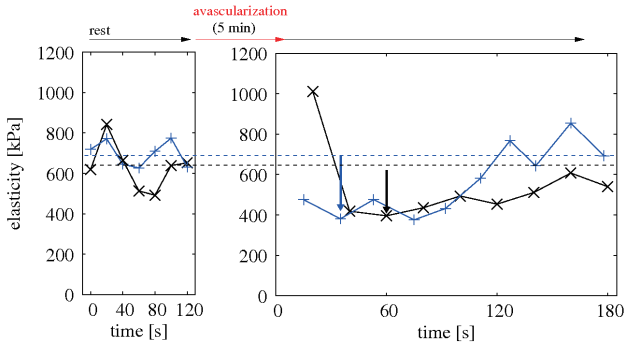

Fig. 4 Transient changes in the static elasticity estimates (avascularization at the upper arm (black) and the forearm (blue)).

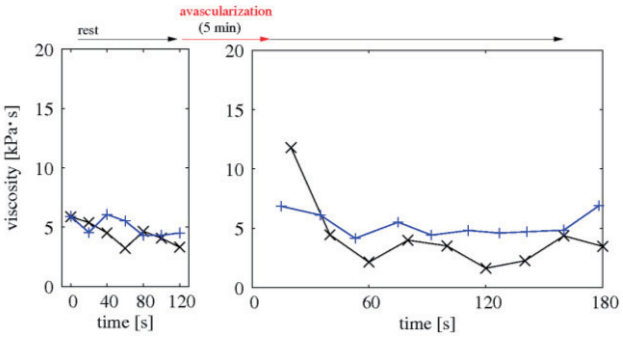

Fig. 5 Transient changes in the viscosity estimates when avascularization was at the upper arm (black) and the forearm (blue).

Figure 4 shows the transient change in static elasticity estimates due to FMD. The static elasticity shows about 40% decrease after avascularization. Decrease in the elasticity by the avascularization measured at the upper arm occurred slower and longer than that measured at the forearm. By avascularization at both parts, the FMD reaction was observed. Considering the stability of the measurement, it is easier to induce avascularization at upper arm than forearm because the upper arm is more distant from the position of the ultrasonic probe and pressure sensors.

Figure 5 shows the transient change in viscosity. The viscosity coefficients measured at rest are almost constant. Just after recirculation, the viscosity coefficient became slightly lower than at rest. In future work, it is necessary to confirm such phenomena reading the change in viscosity due to FMD by measurements of more number of subjects.

4. Conclusion

In the present study, by considering the change in pulse wave velocity during FMD, we showed that the stress-strain relationship could be obtained correctly. In addition, we evaluated the stability in measurement of viscoelastic properties of the radial arterial wall by applying avascularization in upper arm and forearm. As a results, avascularization in the upper arm realized more stable measurement. In future work, the phenomena reading the change in viscoelasticity due to FMD should be further investigated.

References

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