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In Vivo Measurement of Propagation of Myocardial Contraction along Swine Heart Wall

ブタ心筋の収縮伝播特性の *in vivo* 計測

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1. Introduction

Non-invasive identification of ischemia region is valuable for diagnosis of myocardial infarction. Konofagou *et al.* reported that propagation velocity of myocardial contraction in response to electrical stimulation decreases in ischemic region of open-chest canine hearts (normal region: 1.2 m/s, ischemia region: 0.7 m/s)[1)]. For the identification of ischemic region of heart wall, we apply high-accuracy ultrasound measurement to investigate the propagation of myocardial contraction along the heart wall in an open-chest swine around the time of avascularization.

2. Methods

2.1 Measurement of Myocardial Velocity Waveform Originated from Micro Vibration

When ultrasound pulses (center angular frequency $\omega_0 = 2\pi f_0$) are transmitted at the pulse repetition time of ΔT, the phase difference between two succecive received signals $\Delta\theta(x;t)$ is expressed by

$$\Delta\theta(x;t) = \theta(x;t+\Delta T) - \theta(x;t), \qquad (1)$$

where t is the transmit time of the first pulse. Therefore, myocardial micro vibration $v(x;t)$ during the pulse repetition time ΔT is measured by the following equation.

$$v\left(x;t+\frac{\Delta T}{2}\right) = \frac{c_0}{2\Delta T}\frac{\Delta\theta(x;t)}{\omega_0} \qquad (2)$$

In the present study, we calculate a phase difference at the center frequency f_0 by using the phased-tracking method[2)] and estimate the micro vibration velocity at each time step.

2.2 Time Delay Measurement of Myocardial Contraction

In the present study, we calculate the arrival time delay of contraction response at a measurement position $x = x_{\mathrm{M}}$ from the reference position $x = x_{\mathrm{R}}$ using vibration velocity waveform. Delay time of contraction response of a measurement position from the reference position is estimated from the following equation.

$$(f*g)(m) = \frac{\sum_n \big(f(n)-\bar{f}\big)(g(n+m)-\bar{g})}{\sqrt{\sum_n \big(f(n)-\bar{f}\big)^2}\sqrt{\sum_n (g(n+m)-\bar{g})^2}}, \qquad (3)$$

where $f(n) = v(x_{\mathrm{R}}; n\Delta T)$ and $g(n) = v(x_{\mathrm{M}}; n\Delta T)$ are the myocardial velocity waveforms at the reference position and a measuremet position, respectively, $\bar{f}$ and $\bar{g}$ are the average of vibration velocity waveform, and n is the frame number. The delay time is estimated by $\tau = m_{\mathrm{P}}\Delta T$, where $m = m_{\mathrm{P}}$ when the value of Eq. (3) has the maximum.

2.3 Propagation Velocity Estimation of Myocardial Contraction

We estimate the propagation velocity c_s of contraction response in the heart wall by using the average of delay time of each ultrasound beam. We calculated it at intervals of Δz = 50 μm along the depth direction at each beam.

$$\bar{\tau}_i = \sum_{j=0}^{N}\frac{\tau_{ij}}{N}, \qquad (4)$$

where τ_{ij} is the delay time calculated at beam number i and depth $j\Delta z$. N is the number of average. We estimate the propagation velocity of contraction response c_s by applying the least-square method to $(i \cdot \bar{\tau}_i)$ distribution by assuming that acoustic velocity within the region of interest is constant.

3. *In Vivo* Experimental Result

We employed an ultrasound diagnostic equipment (Aloka SSD-6500) with a probe of 3.75 MHz center frequency to acquire RF data of interventricular septum in an open-chest swine heart. In this experiment, the frame rate was 429 Hz and the number of scanning lines was 13 and angle between successive beams was 5.6°. The scanning line closest to cardiac apex side was beam 0. The reference position in normal condition was set in the beam 4 at the depth of 50 mm and that in

ischemic condition was in the beam 5 at the depth of 50 mm.

The left anterior descending artery (LAD) was obstructed manually and measured again after the measurement of normal condition to make a comparison between propagation of myocardial contraction in normal condition and that in ischemic condition.

Figures 2 and 3 show the distribution of the delay time in myocardial contraction response on a B-mode image in normal and ischemic conditions. The delay time increased from apex side to basal side in both normal and ischemic conditions. This result shows the propagation of contraction response from apex side to basal side.

Figure 4 shows $(i, \bar{\tau}_i)$ distribution with respect to lateral position. The propagation velocity in normal condition was 2.17 m/s, and that in ischemic condition was 1.19 m/s. This result shows that myocardial ischemia suppressed the propagation velocity of myocardial contraction.

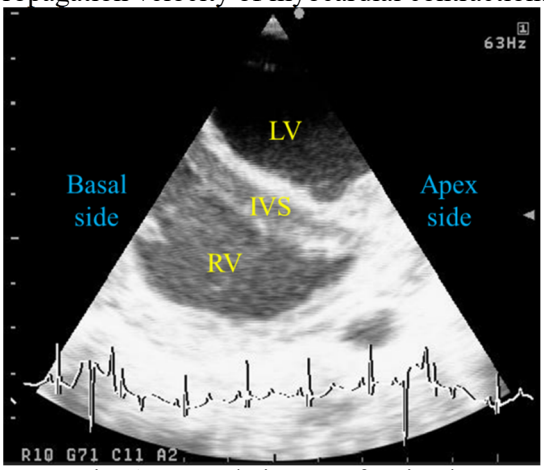

Fig. 1: B-mode image of swine heart.

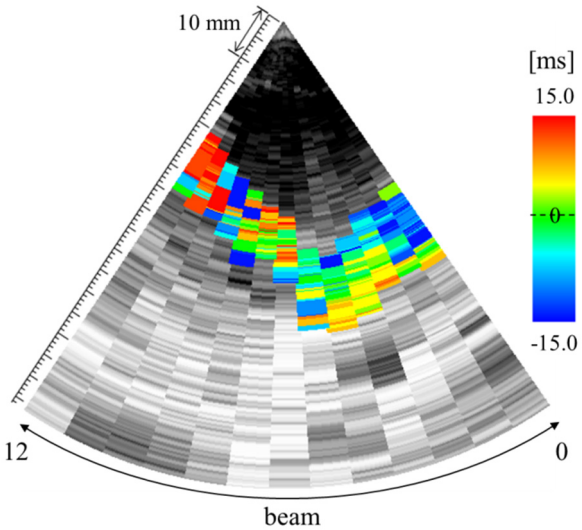

Fig. 2: Delay time distribution of myocardial contraction response in normal condition.

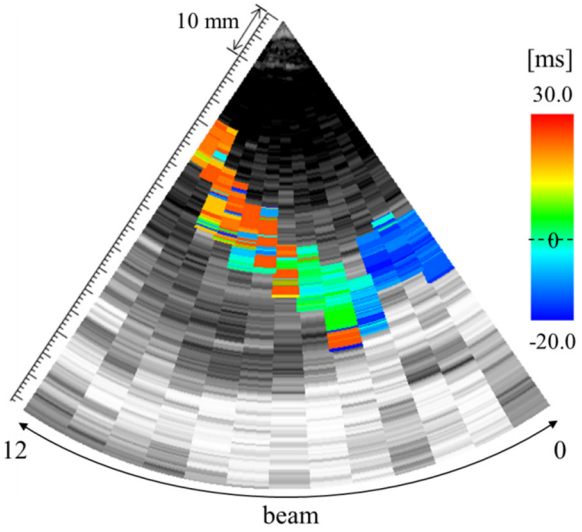

Fig. 3: Delay time distribution of myocardial contraction response in ischemic condition.

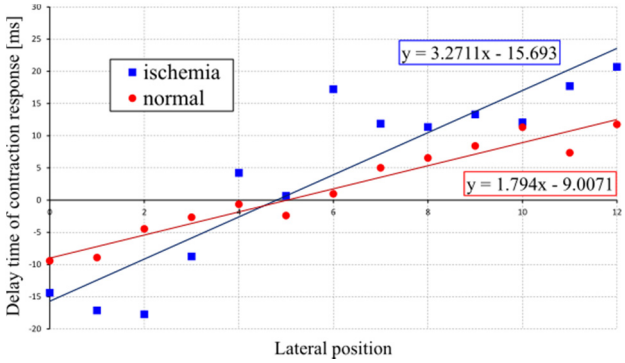

Fig. 4: Average time delays in normal and ischemic conditions.

4. Conclusion

In the present study, we measured the propagation of myocardial contraction along the interventricular septum of an open-chest swine heart around the time of avascularization. We succeeded to visualize the decrease in propagation velocity of myocardial contraction caused by ischemia.

References

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