Ultrasonic Measurement and Analysis of Propagation of Myocardial Contraction Response in Heart Wall

心臓壁における心筋収縮の興奮伝播の超音波計測と解析

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1. Introduction

When ischemia occurs in the cardiac muscle, it is known that abnormality occurs in the contraction function of the myocardium in the ischemic region.¹⁾ Therefore, if we can measure myocardial contractile movement caused by electrical excitation, there is a possibility of early detection of cardiomyocyte abnormalities due to disease. However, the mechanism of the transition process in myocardial contraction has not been fully elucidated.

In the present study, we analyzed the two-dimensional motion in the heart wall using the speckle tracking method for the ultrasonic RF signals measured at the high frame rate and evaluated the contraction characteristic of the myocardium. We estimated the two dimensional velocity of the interventricular septum (IVS) at the time near the R-wave of the electrocardiogram (ECG) and the temporal change of the spatial distribution to evaluate the propagation of myocardial contraction response.

2. Methods

In the present study, two-dimensional (2D) displacement of the heart wall is estimated by block matching method²⁾ using the cross correlation function between ultrasonic RF signals. At the measurement point (m, k) in the heart wall, the lateral velocity component $v_1(m, k; n)$ and axial velocity component $v_d(m, k; n)$ from *n*-th frame to (n + 1)-th frame were determined as follows:

These velocities are expressed by the following equation using the shift $(\widehat{\Delta m}, \widehat{\Delta k})$ that maximizes the cross correlation coefficient between ultrasonic RF signals between two frames.

$$v_l(m,k;n) = \widehat{\Delta m} \delta_l f_{\text{FR}}, \quad (1)$$
$$v_d(m,k;n) = \widehat{\Delta k} \delta_d f_{\text{FR}}, \quad (2)$$

where δ_d is the sampling interval, δ_l is the beam interval, and f_{FR} is the frame rate.

These processes were repeatedly applied to

all the acquired frames to estimate the 2D velocity of the heart wall during one heartbeat. The size of a correlation kernel used for in the IVS is 12.9 mm × 10.2 mm ($\delta_l = 262 \,\mu$ m, $\delta_d = 51.3 \,\mu$ m).

Furthermore, phase components of these velocity waveforms were obtained by applying Hilbert transformation. We determined the equiphase line of the unwrapped phase waveforms by the method of the least squares to estimate the component propagating at velocity c.

3. In Vivo Experimental Result

We acquired RF data of the IVS of a 23-year-old healthy male using an ultrasound diagnostic equipment (Prosound α -10, Aloka, Tokyo, Japan) with a probe of 3.75-MHz center frequency. A high frame rate ($f_{\rm FR} = 860$ Hz) was realized by acquiring RF signals in the parallel beam forming method using plane waves.³⁾ B-mode image of a left long axis cross-section of the heart is shown in Fig. 1.

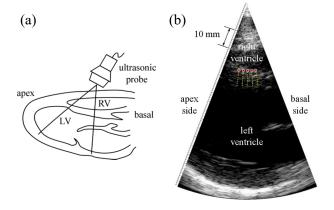


Fig. 1: (a) Left ventricular long axis view (RV: right ventricle, LV: left ventricle). (b) B-mode image in the heart wall of a 23-year-old healthy male.

We estimated the velocity waveforms at 5 points from the basal side to the apex side of the IVS shown in Fig. 1(b). Figure 2 shows the ECG, the phonocardiogram (PCG), the axial velocity waveforms, and the propagation velocity of the contraction response along the lateral direction. Figure 3 shows ECG, PCG, the lateral velocity

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components and the contraction response propagation velocity along the lateral direction.

As shown in Fig. 2 (a), after the time of the Q-wave of the ECG, the axial velocity has changed from the right ventricular side to the left ventricular side, and the transition of the myocardial wall from expansion to contraction was identified. As shown in Fig. 3 (a), the lateral velocity has changed from the basal side to the apical side, and the transition of the myocardial wall from expansion to contraction was also observed. In both the axial and lateral directions, the contraction response propagates from the basal side to the apical side at a speed of about 2 m/s. This will be caused by the propagation of the electrical excitation with the propagation speed of electrical excitation of $2 - 4 \text{ m/s}^{-4}$.

Furthermore, we determined the propagation speed at each time by analyzing the phases components of the velocity waveforms. As shown in Figs. 2(b) and 3(b), it was confirmed that contraction responses propagated at a speed of approximately 1-5 m/s from the Q-wave of the ECG to the beginning of the first heart sound.

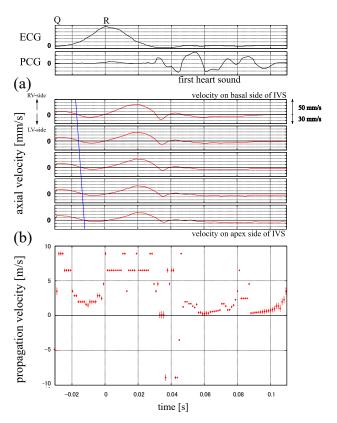


Fig. 2: *In vivo* measurement for the healthy man in Fig. 1. (a) Velocity in the axial direction. (b) Propagation velocity of myocardial contraction.

4. Conclusion

From the two-dimensional velocity of the interventricular septum measured by the speckle tracking method, we observed the motion transiting to the contraction around the time of R-wave. In addition, we observed that the contraction response in the interventricular septum propagated from the basal side to the apical side with high temporal resolution. Furthermore, using the phase components of the velocity waveforms, the propagation speed at each time was determined. From these results, it is suggested that the cardiac contractile function analysis by the proposed method is useful for identifying the mechanism of the transition process in myocardial contraction and for diagnosis of the cardiac function evaluation.

References

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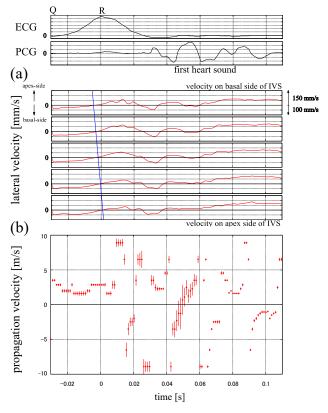


Fig. 3: *In vivo* measurement for the healthy man in Fig. 1. (a) Velocity in the lateral direction. (b) Propagation velocity of myocardial contraction.