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Speeds of Contraction Responses Propagating along Septum at Pre-ejection Period are Different Between Radial and Longitudinal Directions

Hiroshi Kanai
Dept . of Electronic Eng.
Tohoku University
Sendai, Japan
kanai@ecei.tohoku.ac.jp

Shohei Mori
Dept . of Electronic Eng.
Tohoku University
Sendai, Japan
mori@ecei.tohoku.ac.jp

Mototaka Arakawa
Dept . of Biomedical Eng.
Tohoku University
Sendai, Japan
arakawa@ecei.tohoku.ac.jp

***Abstract*—Measuring myocardial contractile movement caused by electrical excitation leads to early detection of abnormalities of cardiomyocytes due to disease. However, the detailed mechanism of the transition process in the myocardium from dilation to contraction at the pre-ejection period is still unclear. In the present study, we acquired ultrasonic RF signals with a high frame rate of 1.16 ms using the parallel beamforming [1] by transmitting a plane wave with a sector probe. Velocity waveform in the 2D direction, the beam direction (the radial direction in the heart wall), and the direction orthogonal to the beam (the longitudinal direction in the heart) were simultaneously estimated by applying the speckle tracking along the heart wall. The cross-correlation coefficient was interpolated so that the spatial resolutions in displacement estimation along the radial and longitudinal directions were increased to 1.0 and 2.2 μm, respectively. We then detected the propagation speeds of contraction response due to electrical excitation along the radial and longitudinal directions at the pre-ejection period. The radial component of 2D velocity waveforms was measured at 112 points along the septum around the R wave of ECG. The velocity component to RV (LV expansion) and that of LV (contraction) were obtained. In the period around R wave, two components propagated along the septum from the basal to apical sides with speeds of 3 m/s and 6 m/s. On the other hand, for the longitudinal component, three components propagated with speeds of 1 m/s, 3 m/s and 12 m/s. Though these all velocity components were simultaneously measured for the same points in the septum, the situations of 2D velocity waveforms were quite different, which would be useful for understanding what occurs at the pre-ejection period.**

Keywords—propagation of myocardial contraction, pre-ejection period

I. INTRODUCTION

The cardiac disease has been already diagnosed using ultrasound by observing the temporal change in morphology and movement of the heart from tomographic images of B-mode and M-mode. Moreover, the ultrasonic Doppler method has been a useful diagnostic tool for blood flow inside the heart. Recently, for quantitative evaluation of diastolic function in the diseased heart, myocardial passive stiffness has been noninvasively measured using the shear wave imaging based on the ultrasound-based ultrafast technique [2] and applied to coronary artery imaging in diastole [3]. In systole, however, the heart wall has complex phenomena, that is, there is the propagation of a few natural mechanical waves and propagation of the myocardial response to the rise of the action potential caused by the underlying electrical stimulation [4, 5]. Therefore, the mechanism of the transition process in the myocardium from dilation to contraction has not been fully understood.

The significance of the noninvasive diagnosis by ultrasonic-based measuring the myocardial response to the propagation of the action potential can be described as follows: When myocardial ischemia occurs in ischemic heart disease, necrosis can be avoided if rapid recovery of blood flow is promptly achieved. Several studies have focused on the propagation of myocardial ischemia [6-7]. The apparent propagation velocity of myocardial contraction caused by the propagation of the electrical excitation decreases in the ischemic region of the canine or porcine hearts in thoracotomy [8-11]. On the other hand, the propagation speed of the contraction response to myocardial electrical stimulation is reported as several m/s [4, 5, 12]. This abnormality of the contractile response to myocardial electrical stimulation has been found not only in ischemic heart disease but also in most typical diseases such as arrhythmic disease, dilated cardiomyopathy, or hypertrophic cardiomyopathy [13].

Therefore, if the decrease in the propagation speed of the contract phenomenon caused by electrical excitation is noninvasively evaluated, regional myocardial damage due to heart disease could be detected noninvasively. To achieve this possibility, however, a breakthrough diagnostic technique is required. That is, since electrical excitation of the left ventricle migrates from the sinoatrial node to the apex of the His bundle and the Purkinje fibers in the left and right bundle branch at a high rate of 1–4 m/s [14], a high temporal resolution is required to capture the propagation caused by electrical excitation.

For this purpose, in the previous study [15], we acquired ultrasonic radio frequency (RF) signals with a high-resolution time of 1.2 ms using the parallel beamforming method [1] by transmitting a plane wave to the interventricular septum (IVS) on the parasternal longitudinal-axis view. The velocity waveforms in the 2D direction (the ultrasonic beam direction: longitudinal direction, and the direction orthogonal to the

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ultrasonic beam: radial direction) of the heart wall were simultaneously estimated by the correlation-based block matching with respect to the ultrasonic RF signals acquired at a high frame rate.

In the present study, the measurement method was applied to the healthy young subjects, from the resultant velocity waveforms in the 2D directions, it was experimentally detected that the speeds of contraction responses propagating along the septum at pre-ejection period are different between radial direction and the longitudinal direction.

II. Methods

A. Displacement estimation method

In the present study, 2D displacement and its velocity waveform of each point set in the heart wall were estimated by the block matching method using the cross-correlation function between ultrasonic RF signals. At the measurement point (m, k) set in the IVS at kth depth along mth direction, the longitudinal velocity component $V_{\text{longitudinal}}(n; m, k)$ and the radial velocity component $V_{\text{radial}}(n; m, k)$ from the nth frame to the (n+1)th frame were determined. These two velocity components are determined by the shift (Δm, Δk) that maximizes the cross-correlation coefficient between ultrasonic RF signals between the succeeding two frames.

This process was repeatedly applied to each of the acquired frames to estimate the 2D velocity waveform of the heart wall during one heartbeat. The size of the employed correlation kernel was 12.9 mm in the IVS along the beam direction and 10.2 mm in the lateral direction (δ_L= 262 μm, δ_R= 51.3 μm). The cross-correlation was interpolated so that the resolutions in the axial and lateral directions are increased to 1.0 and 2.2 μm, respectively.

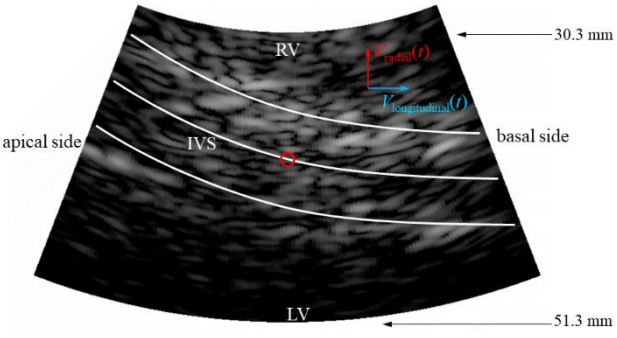

Fig. 1. The B-mode image in the heart wall of a 23-year-old healthy male. The red circles show the analysis points set on the interventricular septal wall. Along each of three lines passing through the IVS from the basal side to the apical side, 112 points were set. For each point, 2D velocity waveforms were measured.

III. Results and Discussion.

A. The motion of the ventricular septal wall

Two-dimensional tracking was applied to 112 points on each of the three lines set in the IVS wall as shown in Fig. 1, and the 2D velocity was estimated as waveform at each point. Figure 2 shows the longitudinal velocity component $V_{\text{longitudinal}}(n; m, k)$ and the radial velocity component $V_{\text{radial}}(n; m, k)$ during -80 ms to +80 ms around R-wave of the electrocardiogram (ECG) for the point at the center of Fig. 1 with an electrocardiogram and a phonocardiogram. As shown in Fig. 2, the IVS wall begins to move in the left ventricular direction (red line of the upper figure) and apical direction (blue line) at the beginning of the systole. However, the movement of the IVS is very complex as shown in Fig. 2 even at the beginning of systole (pre-ejection period).

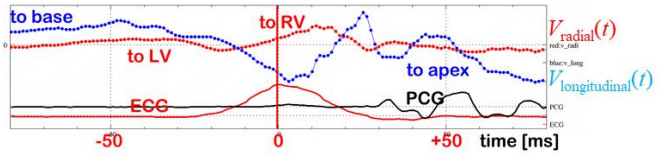

Fig. 2. The radial component $V_{\text{radial}}(t)$ and longitudinal component $V_{\text{longitudinal}}(t)$ of the measured 2D velocity waveform at the analysis point at the center of Fig. 1. ECG: electrocardiogram, PCG: phonocardiogram.

B. Propagation of the myocardial contractile response

Upper figure (a) of Fig. 3 shows the instantaneous spatial distribution of the resultant radial component of 2D velocity waveforms at 112 points along the septum around the R wave of ECG. The velocity component to RV (LV expansion) and that of LV (contraction) were shown in blue and red, respectively. Two components propagated along the septum from the basal to apical sides with speeds of 3 m/s and 6 m/s.

On the other hand, the lower figure (b) of Fig. 3 shows the instantaneous spatial distribution of the resultant longitudinal component of 2D velocity waveforms. Three components propagated with speeds of 1 m/s, 3 m/s and 12 m/s around R wave of ECG.

Though these velocity components were simultaneously measured for the same points, the situations of 2D velocity waveforms were quite different, which suggests that the present results would be useful for understanding what occurs at the pre-ejection period.

For the other two healthy young subjects, similar results were obtained. Therefore, we confirmed that the myocardial wall transited from expansion to contraction at the pre-ejection period. Moreover, there was a slight delay of approximately 4 ms of the myocardial contractile response from the most basal side to the apical side in the ultrasound beam direction and the lateral velocity waveforms. The contraction response propagated at a velocity of approximately several m/s from the basal direction to the apical direction. This corresponds to the propagation velocity of electrical excitation of 1–4 m/s [14].

IV. Conclusions

In the present study, we visualized the 2D velocity of the ventricular septal wall as measured by the speckle tracking method with vectors. We observed detailed movements of the ventricular septal wall near the R wave transitioning from

expansion to contraction. We also confirmed the propagation of the contraction response of the ventricular septal wall from the basal direction to the apical direction by velocity waveforms, which were measured with high temporal resolution. Furthermore, the propagation velocity of the contraction response at each time point was calculated. The contraction response propagates at a rate of approximately 1–4 m/s from the Q wave to the first sound. These results were commonly observed among three healthy males. In these results, however, the speeds of contraction responses propagating along the septum at the pre-ejection period are different between the radial direction and the longitudinal directions. Our results suggest that cardiac contractile function analysis by the proposed method may be useful for evaluating cardiac function.

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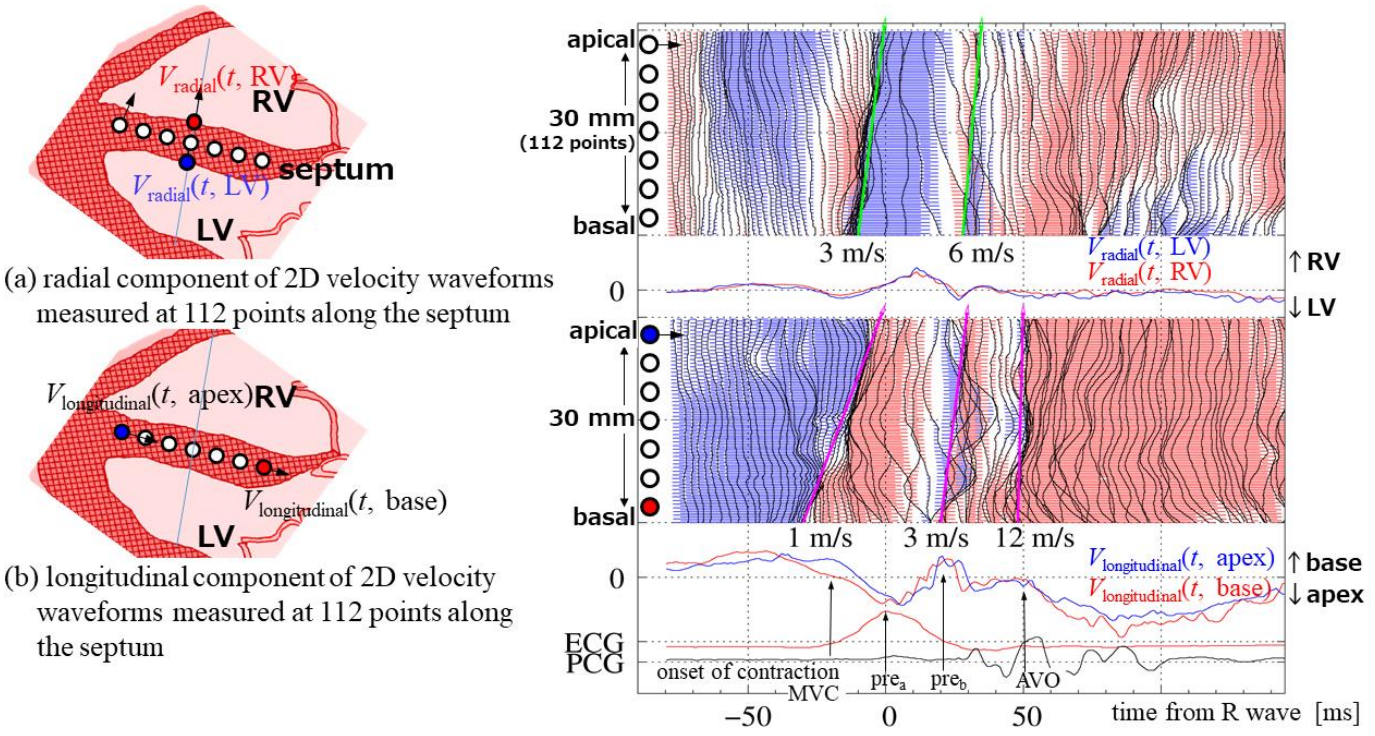

Fig. 3. *In vivo* measurement of propagation velocity for a healthy subject, calculated from (a) the radial component of 2D velocity waveforms measured at 112 points along the septum and (b) the longitudinal component of 2D velocity waveforms measured at 112 points along the septum. (a) The radial component of 2D velocity waveforms was measured at 112 points along the septum around the R wave of ECG. The velocity component to RV (LV expansion) and that of LV (contraction) were obtained. In the period around R wave, two components propagated along the septum from the basal to apical sides with speeds of 3 m/s and 6 m/s. (b) On the other hand, the longitudinal component of 2D velocity waveforms. In the period around R wave, three components propagated with speeds of 1 m/s, 3 m/s and 12 m/s.