Ventricular Excitation Maps Using Tissue Doppler Acceleration Imaging: Potential Clinical Application

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OBJECTIVES
The purpose of this study is to validate the use of tissue Doppler acceleration imaging (TDAI) for evaluation of the onset of ventricular contraction in humans.

BACKGROUND
Tissue Doppler acceleration imaging can display the distribution, direction and value of ventricular acceleration responses to myocardial contraction and electrical excitation.

METHODS
Twenty normal volunteers underwent TDAI testing to determine the normal onset of ventricular acceleration. Two patients with paroxysmal supraventricular tachycardia and 30 patients with permanent pacemakers underwent introduction of esophageal and right ventricular pacing electrodes, respectively, and were studied to visualize the onset of pacer-induced ventricular acceleration. Eight patients with dual atrioventricular (AV) node and 20 patients with Wolff–Parkinson–White (WPW) syndrome underwent TDAI testing to localize the abnormal onset of ventricular acceleration, and the results were compared with those of intracardiac electrophysiology (ICEP) tests.

RESULTS
The normal onset and the onset of dual AV node were localized at the upper interventricular septum (IVS) under the right coronary cusp within 25 ms before the beginning of the R wave in the electrocardiogram (ECG). In all patients in the pacing group, the location and timing of the onset conformed to the positions and timing of electrodes (100%). In patients with WPW syndrome, abnormal onset was localized to portions of the ventricular wall other than the upper IVS at the delta wave or within 25 ms after the delta wave in the ECG. The agreement was 90% (18 of 20) between the abnormal onset and the position of the accessory pathways determined by ICEP testing.

CONCLUSIONS
These results suggest that TDAI is a useful noninvasive method that frequently is successful in visualizing the intramural site of origin of ventricular mechanical contraction. (J Am Coll Cardiol 1999;33:782–7) © 1999 by the American College of Cardiology

Radiofrequency catheter ablation techniques have developed rapidly since 1990 (1,2). They have been found to be efficient for the treatment of sustained supraventricular tachycardia and ventricular arrhythmias. However, a reliable, efficient, noninvasive approach to the intramural localization of abnormal conduction and accessory pathways, such as those in Wolff–Parkinson–White (WPW) syndrome, has not been sufficiently studied. Such a technique could be used to guide the radiofrequency ablation electrode to the proper cardiac chamber, target tissue and could help determine the effectiveness of the ablation procedure. Currently, intracardiac electrophysiology (ICEP) testing and fluoroscopy are used to localize the site of abnormal ven-

tricular stimulation, as in the accessory pathway of WPW syndrome and ventricular arrhythmias. The electrophysiologic technique, however, is invasive, requires radiation exposure and time consumption, and lacks spatial resolution of cardiac structures (3,4). It has been recognized that tissue Doppler imaging can display the distribution, direction and value of myocardial contraction–relaxation energy, velocity and acceleration within the ventricular wall during the cardiac cycle (5–7). The site of initial contraction of a ventricular arrhythmia or in WPW syndrome is recognized as the position of the initial velocity (8). Acceleration is proportional to the change of velocity over time. This parameter is more sensitive to the rapid change of tissue motion than velocity. We hypothesize that tissue Doppler acceleration imaging (TDAI) can be used to detect the onset of myocardial acceleration in response to electrical excitation. There is a need to validate TDAI as a means for evaluating the intramural onset of ventricular mechanical
contraction and to demonstrate its potential clinical applicability.

METHODS

Tissue Doppler Acceleration Imaging

A commercial Acuson XP/10 with TDAI capabilities was used with a V4c trifrequency transducer (2.5, 3.5 and 4.0 MHz). The frame rates used in the real-time TDAI were 40 frames/s with a velocity range of 0.023 to 0.17 m/s. The temporal resolution was 25 ms at the frame rate of 40 frames/s. The zoom mode was used to increase the frame rate. Conventional two-dimensional (2D) gray-scale echocardiography and color flow Doppler imaging were used to display anatomical structures and blood flow for the determination of spatial orientation and recognition of TDAI artifacts. The real-time TDAI was set to record ventricular contraction for ease of displaying the change in the acceleration value. Color-coded display (unidirectional) maps of acceleration distribution and acceleration values on 2D images avoided color confusion. Blue represented the lowest acceleration; red, the highest, and yellow, intermediate. The Doppler frequency shift signal filter, velocity scale and color gain were optimized to increase the signal to noise ratio. Electrocardiographic (ECG) gating was used to control the phase of the cardiac cycle in cine-loop.

Respiration was held at end-expiration to control for respiratory motion artifacts. Artifacts from intracardiac factors (regurgitation, shunt, motion of mitral valve, artificial apparatus, left ventricular band and membrane, arrhythmia, abnormal segment motion) were excluded from the final image analysis by comparison with the baseline images of conventional 2D gray-scale echocardiography, color flow Doppler imaging and TDAI.

All images during the cardiac cycle (before, during and after electrical stimulation in the pacing group and end-diastole and end-systole in the normal and observation groups) were recorded separately by cine-loop and super VHS videotape. Analysis of ventricular acceleration began at end-diastole and continued throughout early systole (at the Q wave, delta wave with WPW syndrome and R wave) to visualize frame by frame acceleration phenomena. Initial ventricular acceleration (i.e., yellow and red) was determined in several cardiac cycles (>5 times) by two experienced physicians before the true onset of ventricular contraction was designated.

Normal Volunteer Group

Twenty volunteers (12 women, 8 men; aged 18 to 30 years) without a history of arrhythmias or heart disease and with normal ECG and echocardiographic findings were studied.

The long-axis and short-axis views of both the left and the right ventricles were observed from end-diastole to early systole to determine the position of onset of ventricular acceleration.

Regional long-axis and short-axis views of the upper interventricular septum (IVS) were observed between P and R waves in the ECG to determine the intramural onset and propagation of ventricular acceleration.

Pacing Group

Esophageal pacing study. Two patients with paroxysmal supraventricular tachycardia (PSVT) (both male, aged 26 and 70 years) were studied.

An esophageal pacing catheter (PES-4 electrophysiologic stimulator) was inserted through the nose into the esophagus. The position of the stimulating electrode was identified by esophageal ECG. The stimulating electrode was placed opposite the left ventricular posterior wall near the atrioventricular (AV) annulus. The stimulating pulse width was set at 10 ms, and the voltage was set at 30 V. A train of 10 pulses was released at one time and repeated 16 times.

The three American Society of Echocardiography standard short-axis views and the two long-axis views (with and without papillary muscle) of the left ventricle were displayed. Each ventricular contraction induced by a train of electrical stimulating pulses was observed frame by frame to localize the position of onset of ventricular acceleration.

Right ventricular pacemaker study. Thirty patients with permanent right ventricular pacemakers were studied (6 women, 24 men; aged 45 to 72 years); 28 of these patients were pacemaker-dependent.

Pacesetter 2040 and Teletronics meta 1206 VVI pacemakers were used with a rate setting of 70 beats/min. The width of the stimulating pulse was 0.35 to 0.50 ms, and the voltage was set to <1.0 V. The electrode of the pacing catheter was placed at the right ventricular wall (apical right ventricular anterior wall in 11 patients, apical right ventricular posterior wall in 13, right ventricular surface of the interventricular septum [IVS] in 3, apical right ventricular lateral wall in 1 and tricuspid annulus in 2).

The pacing catheter was initially localized on long- and short-axis views of the right ventricle. The position of onset of ventricular acceleration was localized from end-diastole to early systole of the pacing cardiac cycle by cine-loop.
Observation Group

Dual AV node group. Eight patients with dual AV node were studied (five women, three men; aged 18 to 36 years). Diagnosis was by esophageal pacing test. The position of initial ventricular acceleration was determined in the long-axis view of the left ventricle and apical four-chamber view at early systole.

Wolff–Parkinson–White syndrome group. Twenty patients with WPW syndrome (diagnosed by ECG) were studied (type A, 15 patients; type B, 5; 12 men, 8 women; aged 19 to 74 years).

The short-axis view of the left ventricle at the mitral valve orifice level and long-axis views were observed to localize the position of abnormal onset of left ventricular acceleration during the delta wave in type A disorder (i.e., the left ventricle is preexcited first). The apical four-chamber view, right ventricular inlet view and short-axis views of the right ventricle were observed to localize the position of abnormal onset of right ventricular acceleration (yellow and red) during the delta wave in type B disorder (i.e., the right ventricle is preexcited first). All patients subsequently had an ICEP test.

RESULTS

Volunteer Group

The position of systolic onset of ventricular acceleration was localized at the anterior upper IVS under the right coronary cusp (RCC) during the R wave in the ECG in all 20 volunteers (Fig. 1).

In regional IVS evaluation, the intramural systolic onset of ventricular acceleration occurred within 25 ms before the beginning of the R wave in the ECG and proceeded from the membranous part to the mid and inferior parts (Fig. 2).

Pacing Group

Esophageal pacing. The position of abnormal systolic onset of left ventricular acceleration was localized at the left ventricular posterior wall near the mitral annulus within 25 ms after the electrical stimulating pulse was initiated. Agreement was 100% between the position of abnormal systolic onset of the left ventricular acceleration and the position of the esophageal pacing electrode (Fig. 3).

Right ventricular pacing. The positions of abnormal systolic onset of right ventricular acceleration were localized at the apical right ventricular lateral wall (one patient), right

Figure 1. Normal case. Long-axis view of the left ventricle. (A) Electrocardiographic gate indicated that the time phase was end-diastole; there was low ventricular acceleration (blue). (B) Electrocardiographic gate indicated that the phase was early systole. The position of normal onset of ventricular acceleration (arrow, red and yellow area) was within the upper interventricular septum beneath the right coronary cusp.

Figure 2. Normal case. Magnified long-axis view of the regional upper interventricular septum. (A) The position of normal onset of ventricular acceleration was within the upper interventricular septum beneath the right coronary cusp (yellow and red area) at early systole. (B) The propagation of ventricular acceleration proceeded from membrane to mid part at early systole.
ventricular posterior wall (13 patients), right ventricular anterior wall (11 patients), right ventricular surface of the IVS (three patients) and tricuspid annulus (two patients) within 25 ms after the electrical stimulating pulse was released. The agreement was 100% between the position of abnormal systolic onset of the right ventricular acceleration and the position of the pacing electrode (Fig. 4).

**Observation Group**

**Dual AV node group.** The position of systolic onset of ventricular acceleration was localized at the anterior-upper IVS under the RCC within 25 ms before the peak of the R wave in the ECG in all eight patients.

**Wolff–Parkinson–White syndrome group.** The positions of abnormal systolic onset of ventricular acceleration were localized separately at the left ventricular posterior wall (six patients), left ventricular lateral wall (three patients), left ventricular anterior wall (six patients), posterior IVS (two patients) and right ventricular posterior wall (three patients) at delta wave or within 25 ms after delta wave in the ECG. The agreement was 90% (18 of 20) with the position of accessory pathways localized from ICEP testing (Fig. 5 and 6).

**DISCUSSION**

Evaluation of ventricular excitation using echocardiography. Since the middle 1970s, M-mode echocardiography has been used first for the localization of the accessory pathways in WPW syndrome. However, because of its inherent limitation (i.e., one-dimensional gray scale technique), the location of the ectopic excitation and preexcitation induced by the accessory pathways could not be exactly defined (9,10). The 2D transthoracic and transesophageal echocardiographic phase analysis technique has been used to locate the accessory pathways in WPW syndrome by evaluating ventricular motion at early systole. However, it is still difficult to localize an intramural focus of the earliest ventricular contraction. The phase angle in phase images presents thickening of the ventricular wall and motion of the ventricular endocardium with lower temporal resolution (16 frames/s) (11,12). Although some previous studies in humans have demonstrated that tissue Doppler velocity imaging can localize most of the accessory pathways in WPW syndrome, even without the classic delta wave or wide QRS complex (8), the accuracy for localization of an intramural focus of myocardial contraction and the local spatiotemporal relationship with electrical excitation remains unclear.

M-mode tissue Doppler velocity imaging as a one-dimensional technique, however, lacks exact spatial anatomical resolution and cannot accurately define the exact spatiotemporal relationship between regional intramural myocardial electrical excitation and mechanical contraction. The tissue Doppler imaging techniques use a Doppler frequency shift gain adjustment and an optimal gate (0.023 to 0.24 m/s) to detect the low frequency and high amplitude Doppler shift signals from myocardium (13).

Electrophysiologic research has shown that the normal onset of ventricular depolarization is at the left side of the upper anterior–mid IVS depolarized by the bundle of His, left and right main bundle branches, during the first 20 ms (14,15). In the normal group, the position of onset of ventricular acceleration is at the upper IVS under the RCC within 25 ms before the peak of the R wave in the ECG. This position and timing conform to the location and timing of the normal onset of ventricular depolarization.
The initial abnormal ventricular exciting positions and timing of the accessory path of WPW syndrome differ from the position and timing of the normal onset of ventricular depolarization and mechanical excitation in normal volunteers. This implies that the TDAI should be helpful for discriminating localization of onset of ventricular contraction.

The results of the pacing study suggest that the location and timing of onset of ventricular contraction of the right and left ventricular walls detected by TDAI confirm the location and timing of external electrical stimulation. Our study also suggests that the TDAI can determine most intramural locations of accessory pathways in WPW syndrome. Cineloop coupled with ECG tracing is useful for observation and analysis to determine the position and timing of onset of ventricular acceleration.

Limitations. The frame rate, angle of the ultrasonic beam and image quality are limitations of the study. A lower frame rate may prevent identification of abnormal onset. In previous tissue Doppler velocity imaging research, it was demonstrated that a frame rate of 36 frames/s was sufficient for the evaluation of onset of ventricular contraction (8). In the WPW syndrome group, in which two cases were misidentified, the accessory pathways were localized at the left ventricular lateral wall. The direction of the left ventricular lateral wall motion was vertical to the ultrasonic beam, and the quality of the image was poor; consequently, the onset and propagation of ventricular acceleration could not be accurately presented. Image quality is also a major factor in the detection of onset and propagation of ventricular excitation. In the dual AV node group, all the onsets of ventricular acceleration were localized at the anterior upper IVS and overlaid the position of normal onset of ventricular acceleration for the same ventricular electrical conductive inlet as that in the normal case. Thus, TDAI cannot differentiate the first excited position of dual AV nodes from that of single AV nodes. The heart translation and rotation during the earliest systole are induced by the initial ventricular contraction at the position of onset (13). Thus, the observation of onset of ventricular contraction cannot be affected by the heart motion.

Technologic improvements. According to the formula of acceleration, it is necessary to improve the capability of the ultrasound system for detecting low velocity with high velocity resolution and to increase the frame rate for obtaining high resolution of time and acceleration. Quality of the TDAI image is very important for localization of the first, smallest excited site within the ventricular wall. Some ultrasound techniques that produce images of high quality (e.g., transesophageal and intracardiac ultrasound techniques) should be helpful. Detection of the accessory path of shading in the preexcited syndrome by TDAI is still
unsuccessful. It should be useful for this kind of detection by shortening the propagation of the accessory path and delaying the propagation of the AV node to induce the forward propagation of the accessory path with esophageal or atrial pacing techniques and some blocking drugs. Adenosine has been shown to increase the sensitivity (65%) for detection of the first contraction site with transesophageal echocardiographic phase imaging (12).

Conclusions. The TDAI technique is useful, clinically feasible and safe for determination of the onset of mechanical contraction in the ventricular myocardium. It should have a significant effect on diagnosis and management in patients with WPW syndrome.

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